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OF

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SESSION 1891-92.

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CONTENTS.

Inaugural Address of Session 1891-92. By JOSEPH EWART, M.D., F.R.C.P. - - - - - - 1

Famine: its Effects and Relief. By Surgeon-General Sir WILLIAM MOORE, K.C.I.E., Q.H.P. - - - - - 27

What is Efficient Vaccination? By Brigade-Surgeon ROBERT PRINGLE, M.D.Edin., late Sanitary Department, H.M.'s Bengal Army - 47

Is Influenza in Man and in Animals Ætiologically Distinct? By RICHARD SISLEY, M.D., M.R.C.P. - - - - 60

Bacteriology of Vaccine Lymph. By S. MONCKTON COPEMAN, M.A., M.D.(Cantab.), D.P.H. - - - - - 70

On the Manifestations of Cholera in Ships at Sea, and their Connections with Outbreaks on Land. By ROBERT LAWSON, LL.D., Q.H.P., Inspector-General of Hospitals - - - - 81

The Seasonal Prevalence of Enteric Fever. (Abstract.) By LOUIS PARKES, M.D.Lond., D.P.H., Lecturer on Public Health at St. George's Hospital, Medical Officer of Health for Chelsea - 108

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1888. Abbate Pacha, Cairo.
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1888. Amado, J. J. da Siloa, M.D., Lisbon.
1888. Bacelli, Guido, M.D., Rome.
1888. Billings, J. S., M.D., War Dept., Surg.-Gen.'s Office, Washington.
1888. Brouardel, S., M.D., 195, Boulevard St. Germain, Paris.
1888. Carsten, B., M.D., The Hague.
1888. Colin, Léon, M.D., 17, Boulevard St. Germain, Paris.
1868. Guipon, D. J., M.D., Laon Aisne, France.
C. 1888. Hirsch, A., M.D., 113, Pottsdamer Strasse, Villa 2, Berlin.
1885. Humphreys, Noel A., General Register Office, Somerset House, W.C.
1888. Hunt, Ezra M., M.D., Trenton, New Jersey, U.S.A.
1888. Koch, R., M.D., 36, Klosterstrasse, Berlin.
1888. Larrey, F. H., M.D., 91, Rue de Lille, Paris.
C. 1888. MacLaurin, H. A., M.D., Macquarie Street, Sydney, N.S.W.
1888. Meijer, van Overbeck de, M.D., Utrecht.
C. 1888. Moleschott, J., M.D., Rome.
1888. Pettenkofer, Max von, M.D., Munich.
1888. Proust, A., M.D., 9, Boulevard Malesherbes, Paris.
1852. Richardson, B.W., M.D., F.R.S., 25, Manchester Square, W.
1850. Simon, Sir John, K.C.B., D.C.L., F.R.S., 40, Kensington Square, W.

* Members are requested to inform the Secretaries of any corrections when necessary.

Elected

c. 1883. Sonsino, Prospero, M.D., 11, Via San Lorenzo, Pisa.
1863. Stark, J., M.D., Bridge of Allen, Stirlingshire, N.B.
1888. Sternberg, Geo. M., M.D., Johns Hopkins University, Baltimore, U.S.A.
1888. Stokvis, B. J., M.D., 26, Sarphatstraat, Amsterdam.
1889. Tholozan, J. Désiré, M.D., Teheran, Phys. to H.M. the Shah.
1888. Vallin, E., M.D., 5, Avenue de Messine, Paris.

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1878. Airy, Hubert, M.D., Local Government Board, Whitehall, S.W.
1891. Anderson, A. J., M.D., Blackpool.
1882. Armstrong, Henry E., D. Hy., Town Hall, Newcastle-on-Tyne.
1888. Ashby, Alfred, M.B., Town Hall, Reading, Berks.
1884. Atkinson, F. E., Whitefriars, Settle, Yorkshire.
1882. Barry, F. W., M.D., Local Government Board, Whitehall, S.W.
1889. Beardmore, G. R., Warwick House, Upper Street, Islington, N.
1889. Boobbyer, Philip, M.B., Municipal Offices, Nottingham.
1891. Braga, J. F., Glen Villa, Sunbury-on-Thames.
1889. Brock, J. H. E., M.D., 115, Adelaide Road, Hampstead, N.W.
1891. Bruce, R. M., Western District Hospital, Fulham.
1870. Buchanan, Sir George, M.D., F.R.S., 27, Woburn Place, W.C. (P. 1881-83. T. 1871-81.)
1889. Bulstrode, H. T., M.D., Hon. Secretary, Local Government Board, Whitehall, S.W.
1887. Butterfield, Harris, Brynmawr, Sevenoaks, Kent.
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1885. Cornish, W. R., Surg.-Gen., C.I.E., 8, Cresswell Gardens, South Kensington, S.W.
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1879. Cory, Robert, M.D., 73, Lambeth Palace Road, S.E.
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1879. Ewart, Joseph, M.D., Dep. Surg.-Gen. Ind. Med. Dept. (*retired*), Montpelier Hall, Brighton.
1875. Fayrer, Sir Joseph, K.C.S.I., M.D., LL.D., F.R.S., Surg.-Gen. Ind. Med. Dept. (*retired*), 53, Wimpole Street, W. (P. 1879-81.)
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1891. Matthews, C. E., M.B., South-Eastern District Hospital, New Cross.
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1883. Paget, Charles E., Town Hall, Salford. (S. 1883-1890.)
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1883. Parsons, H. Franklin, M.D., Local Government Board, Whitehall, S.W.
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1873. Saunders, C.E., M.D., County Asylum, Hayward's Heath, Sussex.

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1878. Smee, A. H., The Grange, Hackbridge, Surrey.
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1888. Thomson, Theodore, M.D., Local Government Board, S.W.
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1880. Condon, J. H., M.D., Surg.-Major Ind. Med. Dept., Cawnpore, N.W.P. India.
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1882. Fasken, William A. D., M.D., Surg. Ind. Med. Dept., Messrs. Grindley and Co., 55, Parliament Street, S.W.
1884. Faulkner, A. S., Surg. Bombay Army, Messrs. Grindlay and Co., 55, Parliament Street, S.W.
1888. Fosbroke, G. H., M.D., Stratford-on-Avon.
1875. Fox, J. Makinson, M.D., The Grove, Lymm, Cheshire.
1883. Furnell, M. C., M.D., Surg.-Gen., Shearman's Gardens, Madras.
1889. Gipps, A. G. P., Royal Hospital, Haslar, Gosport.
1885. Gresswell, D. A., B.A., M.B., Victoria, New South Wales.
1852. Hardwicke, Junius, Clifton Lodge, Rotherham.
1876. Hardwicke, H. Junius, M.D., Burton Lodge, Wostenholm Road, Sheffield.
1872. Harris, Henry, M.D., Trengweath, Redruth, Cornwall.
1880. Harrison, R. Charlton, M.D., 13, Sandringham Gardens, Ealing, W.
1855. Haviland, Alfred, International Club, Trafalgar Square, W.C.
1881. Henderson, G. C., M.D., Kingston, Jamaica.
1882. Herring, John F., St. Mary's House, Atherstone, Warwickshire.
1875. Hime, T. W., M.D., 54, Horton Road, Bradford, Yorkshire.
1875. Home, Sir Anthony D., K.C.B., V.C., M.D., Surg.-Gen., India.
1887. Hunt, J. P., Sheerness Hospital, Kent.
1881. Irwin, Chamney Graves, M.D., Dep.-Surg.-Gen., Edinburgh, N.B.
1855. Jacobson, T. E., Sleaford, Lincolnshire.
1889. James, A. W., Tolleshunt D'Arcy, Kelvedon.
1888. Karkeek, P. G., Isca, Torquay, Devon.
1883. Kirker, Gilbert, R.N., M.D., R. N. Hosp., Gosport.
1879. Lilburne, James, M.D., Dep. Inspec.-Gen., Duncrivie House, Milnathort, N.B.
1886. Lingard, A., Poonah, India.

Elected
1873. Little, James, M.D., 14, Stephen's Green North, Dublin.
1885. Lloyd, W. Harris, M.D., Dep. Inspec.-Gen. (*gone abroad*).
1890. Luard, H. B., Surgeon-Captain.
1881. Lyons, R. T., M.D., Surgeon-Major, Rampart Barracks, Fort William, Calcutta.
1885. McLeod, K., Brigade-Surgeon Lieut.-Col., M.D., LL.D.
1867. Marshall, John, 13, Liverpool Street, Dover.
1860. May, George, jun., 43, Castle Street, Reading, Berks.
1890. M'Neill, R., M.D., Argyll County Council, Oban, N.B.
1881. Oldham, Chas., Brigade Surg. Ind. Med. Dept., Shurdington Road, Cheltenham.
1884. Ray, George, Surg.-Gen., 76, Jermyn Street, S.W.
1880. Reid, Sir J. Watt, K.C.B., M.D., R.N.
1874. Reid, Walter, M.D., Fleet-Surgeon, Royal Naval Hospital, Haslar.
1866. Robertson, James, M.D., Alfred Place, Melbourne.
1863. Rowe, T. Smith, M.D., Cecil Street, Margate.
1879. Ross, J. T. C., C.I.E., Surgeon-General Ind. Med. Dept., The Grove, Ryde, Isle of Wight.
1881. Russell, E. G., M.B., B.Sc., Surg. Ind. Med. Dept., Gauhàti, Assam, India.
1859. Sanderson, J. Burdon, M.D., F.R.S., 64, Banbury Road, Oxford.
1858. Scratchly, G., New Orleans, U.S.A.
1884. Simpson, W. J., M.D., Bengal Club, Calcutta.
1888. Smith, F., Montreal, Cargate Avenue, Aldershot.
1867. Smith, R. Wagstaff, Mount Rundell, Harbourne, Birmingham.
1854. Spinks, C. N., Sankey Street, Warrington.
1889. Street, A. F., M.D., Westgate-on-Sea.
1885. Sturridge, P. F., Kendal, Westmorland.
1880. Tatham, John, M.D., Town Hall, Manchester.
1853. Todd, J. George, Evenwood, West Auckland, Durham.
1885. Tomes, Arthur, Surg. Ind. Med. Service, Bengal.
1873. Townsend, Stephen Chapman, C.B., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner for Central India.
1879. Watson, Alexander, M.D., R.N., Dep. Insp.-Gen., Army and Navy Club.
1880. Whishaw, J. C., M.D., Surg.-Major Ind. Med. Dept., Lucknow.
1880. White, J. Berry, M.D., Surg.-Major Ind. Med. Dept. (*address not known*).
1854. Wiblin, John, Southampton.
1875. Wortabet, John, M.D., St. John's Hospital, Beyrout.

Corresponding Members.

1875. Arkhangelsky, D., M.D., St. Petersburg, Russia.
1852. Barker, R. H., Hungerford, Berks.
1852. Bartolomé, Mariano Martin de, 334, Glossop Road, Sheffield.
1856. Barton, E. H., New Orleans, U.S.A.
1853. Beneke, F. W., Marburg, Germany.
1860. Brionde, A. de, Haute Loire, France.
1851. Cartledge, Benjamin, Vet. Surg., Sheffield.
1871. Christie, James, M.D., Zanzibar.
1875. Colville, W. H., Surg.-Major, Bagdad.
1851. Cooke, R. T. E. B., The Haven, Esplanade Gardens, Scarborough.

Elected

1856. Cox, Wm. Isidore, Hawkesbury-Upton, Chippenham, Gloucestershire.
1871. Cuningham, J. M., M.D., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner of Govt. of India.
1860. Curtis, Josiah, Boston, U.S.A.
1878. Delmas, Louis H., M.D., P.O. Box 524, Havana, Cuba.
1853. Dempster, Charles Carroll, M.D., Dept. Insp.-Gen. (*retired*).
1867. Dickson, E. Dalziel, M.D., Physician to British Embassy, Constantinople.
1851. Galland, —, M.D., Malta.
1863. Hall, E. Swarbreck, 58, Campbell Street, Hobart Town, Tasmania.
1892. Hauser, Philip, M.D., Madrid.
1852. Hebra, Ferdinand, M.D., Vienna.
1889. Hewitt, Red Wing, Minnesota, U.S.A.
1853. Hirsch, August, M.D., 113, Pottsdamer Strasse, Villa 2, Berlin.
1851. Hoskins, S. E., Guernsey.
1886. Janssen, —, M.D., Inspecteur en Chef d'Hygiène, Brussels.
1851. Kellay, —, M.D., Beyrout, Syria.
1881. Kynsey, W. R., Colombo, Ceylon.
1857. Leas, Jose Fernandes da Silva, Cape de Verde.
1875. Lent, —, M.D., Cologne.
1886. Le Roi de Méricourt, L., M.D., 5, Rue Cambacères, Paris.
1851. Litt, W., Veterinary Surgeon, Shrewsbury, Salop.
1857. Lowtzoff, —, St. Petersburg, Russia.
1887. McLaurin, H. A., M.D., Sydney, New South Wales.
1870. Mallet, —, M.D., Warsaw.
1886. Moleschott, Jacob, M.D., Rome.
1851. Monzon, Rafael, Barbacaos, New Grenada.
1853. Neumann, —, M.D., Berlin.
1872. Poggio, Don Ramon Hernandez, Cuba.
1854. Purple, S. S., M.D., New York, U.S.A.
1886. Rochard, G., M.D., Paris.
1851. Sankey, F. F., Malta.
1879. Sarell, Richard, M.D., Pera and Therapia, Constantinople.
1873. Schleisner, —, M.D., Copenhagen.
1874. Schlimmel, —, M.D., Teheran.
1852. Schürmayer, Professor, Heidelberg, Germany.
1851. Sharpe, Richard, Beverley Road, Hull.
1853. Sigmund, C. L., Vienna.
1886. Sonsino, Prospero, 11, Via San Lorenzo, Pisa.
1851. Spooner, W. C., Vet. Surg., Southampton.
1876. Sternberg, Geo. M., M.D., United States Army, Port Point, San Jose, San Francisco, California.
1860. Taylor, H. Sharp, 15, Quarry Street, Guildford.
1855. Tholozan, J. Désiré, M.D., Teheran.
1888. Thompson, Ashburton, M.D., Sydney, New South Wales.
1851. Tribes, E., Nismes, France.
1855. Tribuchet, Adolphe, Paris.
1853. Valerj, —, M.D., Rome.
1855. Virchow, Rudolf, Professor, 10, Schellings Strasse, Berlin.
1856. Von Iffland, —, Beaufort, New Quebec.
1853. Watson, John, M.D., Jamaica.
1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.

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INAUGURAL ADDRESS OF SESSION 1891–92.

By JOSEPH EWART, M.D., F.R.C.P., J.P., Mayor of Brighton.

(*Read:* 18*th November* 1891.)

Gentlemen,—I have first to thank you for your kindness in re-electing me to the honoured office of President of your Society for the year 1891-92. The work of the past session has been well sustained. The subjects selected for discussion have been of great interest; in the aggregate they will form a valuable addition to our *Transactions*. It remains for me to express a hope that the papers submitted during the present session will be equally attractive and useful.

In my former address, I gave a brief review of some recent sanitary legislation, enacted with a view to aid local authorities in dealing with avoidable and preventable disease. It was demonstrated that this legislation was hygienically and fiscally a long way short of meeting the pressing necessities of the times in which we live. The permissive character of a good deal of it was found to be highly objectionable. The cost of providing healthy homes for the people is still rendered forbidding by the dominating existence of false and old-fashioned notions regarding the sacredness even of house property, which has been condemned by the Sanitary Authorities as unfit for human habitation, and by the multiplication of legal formalities and procedure, etc., which might easily have been made more simple and economical. Fresh sources of municipal revenue might have been tapped without any injustice. Thus, with a substantial reduction of the initial expense of purchase incurred in carrying out the provisions of the "Housing of the Working Classes Act, 1890", and the addition of new to the old resources to cover the expenditure involved, the clearing, re-modelling, and re-building of the pestilential slums, insanitary dwellings, obstructive

buildings, and working-class lodging-houses in large towns, would be greatly facilitated. As things have been left, the progress of sanitation must continue to be slow and halting in those unhealthy areas where the vital interests of the community urgently demand that it should be promoted with the greatest promptitude and expedition.

The Public Health (London) Act passed at the end of last session comes into operation on the 1st January 1892. In the main, it is an application of the principles of the Public Health Act of 1875 to the metropolis, with many important amending improvements. It is a consolidation and codification of sixteen Acts, including the greater part of the sanitary law relating to London. Some few illustrations may be alluded to:

1. It has given increased powers to the Vestries, now known as Sanitary Authorities, and authorised to make bye-laws which they will have to enforce.

2. Section 29 imposes the responsibility upon the Sanitary Authority of keeping the foot-ways as well as the streets clean. Failure may be followed by the infliction of pecuniary penalties.

3. Section 1 says: "It shall be the duty of every Sanitary Authority to cause to be made from time to time inspection of their district, with a view to ascertain what nuisances exist, calling for abatement under the powers of the Act, and to enforce the provisions of this Act for the purpose of abating the same, and otherwise to put in force the powers vested in them relating to public health and local government, so as to secure the proper sanitary condition of all premises within the district." Does this mean house-to-house inspection? To some it may seem so. But it would have been better had the express words been used. Does it give the power of entry? It certainly does so inferentially; but why has it not been stated in positive terms? Why this half-heartedness and timidity in matters of such extreme importance? It is true that the 40th section gives "a power of entry quite irrespective of complaint for special purposes". Thus: "The Sanitary Authorities may examine any of the following works, that is to say, any water-closet, earth-closet, privy, ashpit or cesspool, and any water-supply, sink, trap, syphon, pipe, or other works or apparatus connected therewith, upon any premises within their district, and for that purpose, or for the purpose of ascertaining the course of a drain, may at all reasonable times by day, after twenty-four hours' notice has been served on the occupier of the premises, or if they are un-

occupied, on the owner, or in case of emergency without notice, enter on any premises, and cause the ground to be opened in any place they think fit, doing as little damage as may be." This is eminently satisfactory so far as it goes; but it seems to me that it does not go so far as to confer a general power of entry, which is absolutely necessary to make a house-to-house inspection as effective as it should be.

4. In the Public Health Act (1875), Sect. 91, the definition, "a nuisance, or injurious to health", is, in Sec. 2 of the Act under review, extended to "a nuisance, or injurious or dangerous to health". In short, it is not now necessary to show or prove that premises complained of are, or have been injurious; it is sufficient to show that, in their insanitary condition, they must be dangerous to health. The procedure is the same under both Acts, by summary proceedings before the magistrates. But in the London Act appeals are discouraged. "A person may appeal against a nuisance order, provided the order is one for prohibition, or for closing premises, or for the execution of structural works; but if he lose the appeal he has to satisfy the Court that there was substantial ground for the appeal, and that it was not brought for the purpose of delay, otherwise he is liable to a fine of 20*s*. a day."

5. The Sanitary Authority, on the certificate of a Medical Officer of Health or a Sanitary Inspector, are authorised by the Act to require that "workshops shall be lime-washed, cleansed or purified, if this is necessary for the health of the persons employed". In the event of the owner or occupier of the offending premises failing to carry out the order within the time specified, the Sanitary Authority may execute the work itself, and charge the cost of the same to the owner or occupier. "Similar provisions are to be taken as to offensive trades, and power may be exercised for the supply by the Sanitary Authority of public lavatories, ash-pits and conveniences."

6. Sect. 22 empowers the County Council to take proceedings against a Sanitary Authority for neglecting to remove dust, and to abate "possible nuisance arising from dust wharfs, destructors or other methods of disposal". It is the duty of the Sanitary Authority to remove house-refuse at proper periods. "If, forty-eight hours after written notice to the Authority by the occupier, the Sanitary Authority" fail without reasonable cause "to remove the dust, the Authority may be fined £20" (Public Health Act, 1891, p. 9). No doubt the reasonable cause will be dis-

covered, and, if so, what is the real working value of this clause?—probably *nil.*

7. Sect. 47 (Sub-sec. 4) authorises a Court to affix a notice for a period of twenty-one days to a man's shop who has been twice convicted within twelve months of selling unwholesome food, if the Court at the same time finds that he knowingly and wilfully committed both offences!

8. Before any new house can be inhabited, a certificate must be obtained from the Sanitary Authority that an adequate supply of water has been provided for it.

9. Penalties may now be inflicted for jerry-building or defective plumbing. "If a water-closet or drain is so constructed or repaired as to be a nuisance, or injurious or dangerous to health, the person who undertook or executed such constructional repair shall, unless he shows that such constructional repair was not due to any wilful act or neglect, or default, be liable to a fine not exceeding £20." If he can show that his *employés* are the offenders, the said workmen may be prosecuted and convicted (Sect. 45).

10. Sect. 55 (Sub-sect. 4) provides that a copy of each certificate of inspection of infectious disease (furnished under the Infectious Diseases Notification Act) relating to children going to school, and received by the Health Officers, "must be sent to the head teacher of the school, attended by the patient (if a child), or by any child, who is an inmate of the same house as the patient."

11. Important provisions are made with respect to the occupation of underground rooms (Sec. 96) and the closing of houses, the dealing with defaulting Sanitary Authorities by the County Council (Secs. 100 and 101), the appointment and residence of Health Officers, and the qualification of Sanitary Inspectors after 1st January 1895, who is to be the holder of a certificate granted after examination by a body approved from time to time by the Local Government Board, "or shall have been during three consecutive years preceding 1895 a Sanitary Inspector of a district in London, or of some other urban sanitary district of not less than 30,000 population" (Public Health).

Although the Act contains many new principles which, reduced to practice, would materially improve the sanitary condition of the metropolis, it is doubtful whether, under the present constitution of the vestries, its best and most advanced provisions will be put in force with the desired expedition and loyalty. It perpetuates two cardinal defects which have hitherto impeded or thwarted efficient sanitary

progress in those parts of London where it is most imperatively needed: defects which, until reformed, will continue and uphold a vestry organisation too much interested in keeping things as much as possible as they are, the new Act to the contrary notwithstanding. The Act will not have fair-play so long as these blemishes are allowed to mar and mutilate it. The *first* is the property qualification, and a consequent lobsided electorate unduly favouring the owners of insanitary houses, who and their friends will still be, in the future as they have been in the past, almost all-powerful in declining to carry out the sanitary reforms authorised by law; and the *second* is the imposition upon the occupying ratepayers of the entire cost of executing the sanitary laws, instead of assigning a reasonable share of it to be paid by the offending landlords, which would only be fair and equitable, seeing that they are responsible for "many of the nuisances and structural defects which are injurious and dangerous to the health and lives of the people".

Infectious Disease.—Deep down in the recesses of antiquity the theory of the causation of contagious diseases by living organisms was, to some extent, entertained. In the absence of any means of verifying even the existence of such organisms, it is reasonably fair to assume that this hypothesis, which is in the ascendant at the present day, was based on the clinical experience of these fevers. Thus, the tolerable regularity of their beginning, development, growth, culmination, cessation, recovery, or the reverse; a specific and more or less defined train of symptoms; the spread as if from person to person, and the reappearance of the same phenomena in an endless succession of cases, pointing to the existence of a reproductive process both in the causation and course of the disease, were doubtless noted by the physicians of the olden time. And so, as the centuries rolled by and experience accumulated the theory of a *contagium vivum seu animatum*, or the hypothesis that the causes of infectious diseases consist of living organisms of low degree, was duly formulated. Thus minute organisms were viewed as the causes of many forms of malarious fever; and that the Plague was engendered in the same way was believed long before the discovery of the microscope and infusoria by the Dutch naturalist, Leeuwenhoeck. His demonstration of the spermatozoa in 1677 gave an immense impetus to the general acceptance of the doctrine.

Owing, however, to many ill-founded assertions and

reckless generalisations, the hypothesis fell into disrepute. But the recent improvement and vast increase in the magnifying power of the microscope, the discovery of staining dyes, the cultivation of organisms in suitable media and subsequent experiments with them, etc., have, in the hands of competent investigators, combined to restore this hypothesis to much more than its ancient position in the minds of the great body of the medical profession in all parts of the civilised world.

No man, in our time, has done so much to place this theory on what seems to be a secure basis as the illustrious Frenchman, Louis Pasteur,* the most conspicuous scientific figure of the age. To the members of this Society his life-work will be all the more congenial and attractive, because it shows that he has opened out a new kingdom of interesting organisms, "the infinitely little"; solved many problems previously unexplained; shed, by the lustre of his genius, much light on biology; cleared the way for a rational interpretation of the causes of infectious diseases; unfolded the principle on which the Jennerian practice of vaccination is based; and successfully applied that principle to the prevention of anthrax, chicken-cholera, and hydrophobia. The more his method of discovery is studied, the more vividly does it appear that he possesses in an extraordinary degree "the divine power of distilling from facts their essences, or of extracting from them the principles from which they flow", and that "the mind is the standard of the man". He has questioned, verified, and learned. "He has searched out the secrets of nature by experiment."

First Indications of Vitality.—Arago, in 1811, and Biot, in 1812 and 1818, first pointed out "that a plate of rock crystal, cut perpendicular to the axis of the prism, possessed the power of rotating the plane of polarisation through an angle dependent upon the thickness of the plate and the refrangability of the light" (Tyndall). Biot also proved that there were two kinds of rock crystal, one turning the plane of polarisation to the right, the other to the left. They were accordingly called right and left-handed crystals.

On close examination, it was found that this difference of behaviour was dependent upon the existence of minute facets, "which, in the one class, were ranged along a right-handed, and, in the other, along a left-handed spiral", thus disturbing the symmetry of the hexagonal prism, and of

* See his *Life and Labours*, translated from the French by Lady Claude Hamilton, with Introduction by Professor Tyndall, 1885.

the two terminal pyramids of the crystal, and inferentially indicating a corresponding arrangement of the molecular structure of each species. The chemical composition of both was identical.

When, therefore, Mitscherlich declared the crystalline, chemical, and molecular identity of the tartrates and paratartrates of ammonia and soda, one causing the plane of polarisation to rotate while the other did not, Pasteur must have been prepared to search for facets for an explanation of this difference. He found them first in the tartrates. The neutrality of paratartrates he proved to be due to equal proportions of right-handed and left-handed crystals by a beautiful series of experiments.

Pasteur considers that this rotatory property is the earliest exclusive attribute of life. If really true, it bears hardly against materialism, inasmuch as it points to a clear and definite line of demarcation between dead and living matter, or to an insuperable barrier between the organic and inorganic world. Pasteur denies that the chemist has ever produced in his laboratory a substance which, in solution, can permanently cause the rotation of a polarised beam. He contends that this power belongs alone to derivatives from living things. He looks upon the force or forces which produce this deviation of polarised light to the right or left as the agent or agents of vitality. Such molecules are the progeny of parents who have lived.

Wohler, Jungfleisch, others in France and Germany, and Tyndall in this country, contend that this barrier has been destroyed. Tyndall mentions the fact that, whilst a crystal of quartz produces a very powerful twist on the plane of polarisation, in a state of solution it is quite neutral. Here, he thinks, we need not interpolate new forces, those extant in mineral nature sufficing. He also cites the important experiments of Faraday, who, by passing magnetic or electric currents through inert aqueous solutions, caused at his will, in the twinkling of an eye, deviation or rotation of the plane of polarisation. That therefore the power of rotation of the polarised beam of light is, as still affirmed by Pasteur, a vital phenomenon, does not appear to rest on a very impregnable foundation. "For my part", says Tyndall, "I confess my disbelief in its permanence."

Fermentation.—But Pasteur's view, coupled with what at first sight might seem to be a very trivial circumstance, for ever turned his attention from molecular physics to the domain of physiology and biology. A German manufacturer had made it known that the impure tartrate of lime

of commerce, mixed with organic matters and kept under water in the warmth of summer, fermented and yielded various products. Why, asked Pasteur, should not the right-handed tartrate of ammonia ferment in a similar manner?

The question was soon answered by convincing experiments. 1. He dissolved some pure crystalline salt in water with some albuminoid matter, one gramme to 100 grammes of the tartrate, and placed the clear and limpid solution in a warm chamber. It soon became opaque or turbid. It fermented. The microscope showed that the turbidity of the liquid was due to the presence of a small organism. 2. He applied the same process to the tartrate of ammonia; fermentation ensued, and the same organism was deposited. A solution of the paratartrate is neutral, that is, it shows no deviation of a polarised beam of light; because it is composed of an equal proportion of right- and left-handed tartrate. After the fermentation had ceased, Pasteur applied the polariscope, and found that the beam was turned to the left. He evaporated it, and added to the residue its own volume of alcohol, and from this he obtained a beautiful crystallisation of left-handed tartrate of ammonia. 3. He next proved that mildew could thrive and multiply in a purely mineral soil, made up of phosphates of potash and magnesia, and paratartrate of ammonia in solution. He sowed on this some seeds of common mould (*Penicillium glaucum*). In a short time the left-handed acid appeared, and he knew that this could only happen after an equal quantity of the right-handed acid had been decomposed.

Now what did these splendid and unique experiments show? In the first, the small organism acted as a living ferment, multiplying and subsisting upon the carbon of the right-handed tartaric acid of the tartrate of ammonia until it had become exhausted. In the second and third experiments, the solution of the paratartrate does not turn the plane of polarisation to the right or left. But as the fermentation advanced the equilibrium became disturbed; rotation to the left was noticed, and went on until it reached its maximum, when all the right-handed tartrate disappeared, leaving nothing but left-handed acid behind. The organism found the right-handed tartrate most digestible, and, in consuming its carbon in preference to that of the left-handed acid, it manifested its power of selecting with precision its own food. Why should this be so, seeing that the chemical composition of the two acids is identical? It can only be attributed to the peculiarity of

molecular structure, which, whilst conferring upon the substance the power of rotating the plane of polarisation to the right, also made a congenial nutritive material for the organism.

Pasteur at the Parting of the Ways.—Here we find Pasteur standing as it were at the parting of the ways, hesitating as to which way he should go. He had accomplished great things in molecular physics, and the temptation to continue to follow this line of investigation must have been almost overruling. By his former masters and present colleagues he was strongly urged to do so. The route had been made plain; so far he had outstripped in the race all his predecessors and contemporaries in the domain of physics and chemistry. Further success awaited him at each step.

An incident occurred which triumphed over his hesitations. He was appointed Dean of the Faculté des Sciences at Lille, where the chief industry is the production of alcohol from beetroot and corn. Fascinated with the strange molecular action of the two tartaric acids in the presence of a microscopic organism, and inspired with the sympathetic instinct of increasing the local influence of his new faculty, he naturally resolved to devote a portion of his lectures to the study of fermentation.

At the threshold of this inquiry he was confronted by two great facts. "All that has lived must die, and all that is dead must be disintegrated, dissolved, or gasified; the elements which are the substratum of life must enter into new cycles of life. If things were otherwise, the matter of organised beings would encumber the surface of the earth, and the law of perpetuity of life would be compromised by the gradual exhaustion of materials."

"One grand phenomenon presides over this vast work—the phenomenon of fermentation. What is the cause of the processes of fermentation and putrefaction? How is the disappearance of the dead body or the fallen plant to be accounted for? What is the explanation of the foaming of the must in the vintage-cask? of dough, which, abandoned to itself, rises and becomes sour? of milk, which curdles? of blood, which putrefies? of the heap of straw, which becomes manure? of the dead leaves and plants embedded in the earth, which transform themselves into soil?"

Theories of Liebig and of Berzelius and Mitscherlich.—Liebig had revived the well-known theory that ferments were simply nitrogenous matter undergoing change in the

presence of the oxygen of the air. Berzelius and Mitscherlich held that the ferment was an albuminoid substance, which operated by its mere presence or contact, and which took nothing from, and added nothing to, the fermentable matter. The force employed was termed catalytic.

At the beginning of the century, Gay Lussac, in examining Appert's process of preserving animal and vegetable substances by hermetically sealing them and subsequently exposing them to heat, had observed that, on being opened, decomposition soon ensued; and this was ascribed to the disintegrating action of the oxygen of the air. This fitted in, either to the theory revived by Liebig, or that of Berzelius and Mitscherlich.

Overthrow of the Theories of Liebig and Berzelius.—Cagniard Latour in France, and Schwann in Germany, simultaneously repeating the observations of the naturalist Leeuwenhoeck, saw the torula cells of yeast multiplying themselves by a budding process, and the former formed the conception that possibly the fermentation of sugar was connected with the act of cellular vegetation. The great chemist, M. Dumas, also entertained a similar notion. But, as in no other kind of fermentation had any organised being been found, the theory was not pressed. It remained for Pasteur to supply the missing links. He discovered the lactic ferment, consisting of small rods nipped or compressed at the centre, about the thousandth part of a millimètre in diameter multiplying by fission. He isolated these organisms, and cultivated them in suitable media. Pasteur made two experiments which finally overthrew the theories of Liebig and Berzelius: (1) He introduced in a pure solution of sugar a little crystallisable salt of ammonia, some phosphates of potash and magnesia, and sowed in this medium a few cells of yeast. Fermentation rapidly followed. (2) He next mixed with some water, sweetened with sugar, a little salt of ammonia, some alkaline and earthy phosphates and pure carbonate of lime obtained by precipitation. At the end of twenty-four hours the liquid began to get turbid and to emit gas. The fermentation continued for some days. In this case the germs of the lactic ferment which was deposited had been derived from the dust adhering to the substances employed in the formation of the mixture, or to the vessel used, or from the surrounding air.

Living-ferment Hypothesis of Latour and Pasteur reduced to a Fixed Law.—The contact or catalytic theory of Berzelius, and the communicated motion theory of Liebig

and his predecessors, were thus conclusively condemned, whilst the living-ferment hypothesis of Latour and of Pasteur was reduced to a fixed law, or scientific principle. The catalytic or contact theory had no longer any intelligible meaning, since the organised ferment obtained its carbon, etc., from the fermentable material; the communicated molecular motion theory of Liebig, originating in a nitrogenised albuminoid substance, had no better claim, since such matter had been discarded. The torula, in the first case, assimilated and digested whatever it required of the mineral and organic elements for its own sustenance and the reproduction of the species, the chief changes being the formation of alcohol and carbonic acid, and so did the lactic ferment, in the other, among other changes, leading to the production of lactic acid. In these, as, indeed, in every kind of fermentation, the main process took place between the sugar and ferment-germs owing life and the power of multiplication to the nutritive matters of the fermentable substance. Thus, fermentation may be regarded as a process of nutrition in which the organisms manifest in an eminent degree the property of reproduction, development, and growth, in the maintenance of the race, and the building up of their own organisation from sugar and purely mineral elements.

The Ferment of Butyric Acid.—Pasteur next directed his attention to the ferment of butyric acid. He found that it was a vibrio, a little cylindrical rod, rounded at the ends, single or in chains of two or three, or more. It moves by gliding straight, bending or undulating, and multiplies by fission. Like the yeast or lactic organisms, they may be sown in any medium suitable for their nourishment, and then they multiply to infinity. They thrive in ammonia and crystallisable solution, provided these contain fermentable materials, such as sugar, lactic acid, gum, etc. The butyric fermentation proceeds as the organisms multiply. In this, as in other fermentations, although their weight increases, it has been found that this is small in comparison with the amount of butyric acid produced.

The butyric ferments or organisms have, as shown by Pasteur, a peculiarity which distinguishes them from the alcohol and lactic ferments. They not only live without air, but free oxygen is fatal to them, and stops fermentation. Carbonic acid passed into the liquid, where they are multiplying, has no effect in interfering with their vitality or reproduction. But a current of air passed into a portion of the same medium, in the course of two hours causes the vibrios

to fall lifeless to the bottom of the vessel, and at the same time arrests the fermentation dependent upon them.

The newly discovered class of organisms were accordingly called anaérobies, or beings which can live without air; and the others, which, like the larger animals, cannot live without free oxygen, he termed aérobies. "It matters little whether the progress of science makes this vibrio of the butyric ferment a plant or an animal; it is a living organism, endowed with motion, which is a ferment, and which lives without air."

Ferment and Oxygen.—From this discovery Pasteur was led to divine that there might be some hidden relation between the property of being a ferment and the power of living without free oxygen; and in a series of ingeniously devised experiments he showed that the must of grapes and that of beer acted most effectually when the ferment-germ was practically excluded from the air. Beer fermentation is carried on in large and deep vats, and when the process begins the air is soon excluded by the sides of the vats and a deep layer of carbonic acid gas which settles down on the surface. But the torula can live and thrive either with or without free oxygen, as demonstrated by using shallow vessels. The difference is that, in the first instance, the amount of decomposed sugar is less in proportion to the yeast used than in the latter case. In other words, the more free oxygen the yeast consumes, as in shallow vessels, the less is its power as a ferment. On the other hand, the more the life of the ferment is carried on in the entire absence of free oxygen, as in deep vats, the greater is its power of decomposing saccharine matter.

The light which these revelations threw upon the cause of fermentation, coming as they did unexpectedly, produced a strong impression upon the minds of scientific men. M. Dumas, his former teacher, and now his colleague, said one day to M. Pasteur, before the Academy of Sciences: "In these infinitely small organisms you have discovered a third kingdom, the kingdom to which these organisms belong, which, with all the prerogatives of life, do not require air for their existence, and which find the heat that is necessary for them in the chemical decompositions which they set up around them."

Triumphant Reply to Liebig's Objections.—He carried on his remarkable researches into putrefaction, and proved that the *primum movens* of this process resides in vibrios of the same order as those which constitute the butyric ferment. The reason why the use of this term is continued

is justified by the putrid odours of the sulphurous and phosphorous gases given off during the fermentation or decomposition of fibrine, albumen, blood, gelatine, etc. Liebig, in spite of the unerring exactness and precision of Pasteur's experiments, retained and preached the old doctrine of communicated molecular motion. "If", he said, "the fungus is the cause of the destruction of the oak, if the microscopic animalculæ is the cause of the putrefaction of the dead elephant, I would ask in my turn what is the cause which determines the putrefaction of the fungus or of the microscopic animalculæ when life is withdrawn from these two organisms?" Pasteur's querulous countryman, M. Bouillaud, about the same time asked, "Let M. Pasteur tell us here in the presence of the Académie de Médecine what are the ferments of the ferments?" The reply, backed up by a set of decisive experiments, was prompt, characteristic, and indisputable. It may be briefly summarised. A ferment having finished its work and used up all the aliment at its disposal, dies, and becomes an accumulation of dead organic matter. Such, for example, would be a quantity of yeast exposed to the air. As putrefaction goes on, in summer weather, the interior of the mass is pervaded by a host of anaérobic ferments, or vibrios, which continue to do their work of fermentation so long as they are protected from free oxygen. On the surface, in communication with air, mould, bacteria, and other aérobic organisms grow, and, by fixing the oxygen, determine the slow combustion which gasify the mass. Both perish as soon as their nourishment is exhausted, and themselves become the prey of anaérobic or aérobic organisms. Thus the ferments of ferments are simply ferments. So that from fermentation to fermentation, putrefaction to putrefaction, combustion to combustion, the organic mass of yeast finds itself reduced to a collection of germs, and "in the final analysis they represent life in its eternal form, for life is the germ, and the germ is the life."

Anaérobic and Aérobic Organisms, the Masters of the World.—In no department of animated nature is the design of the Creator more strikingly developed than in the life-history and work of these anaérobic and aérobic germs and organisms. As all animals and vegetables—all that have possessed life—must die and become metamorphosed, the absolute destruction of vitality, or *vis vitæ*, is arrested by them, and life thus preserved is transferred to other microbes, and the perpetuation of life ensured. We see what would happen if the climate were continued at a

temperature so low that these organisms could not live. With their death, decomposition or putrefaction is arrested, as exemplified in antediluvian elephants found in the Polar regions of ice, frost, and snow. Such facts illustrate the important part which they play, and show that they are virtually the "masters of the world". What would happen if their work were suppressed? Encumbered with organic matter, "the globe would soon become uninhabitable."

Acetic Fermentation.—Pasteur next engaged in an inquiry into the process of acetic fermentation, and, in a series of experiments, proved that it was due to the presence of a minute microscopic fungus, the *Mycoderma aceti.* Vinegar is made from wine. This cryptogam has the property of condensing large quantities of oxygen, and fixing it upon alcohol, transforming it into acetic acid. Whilst doing this, it feeds and multiplies upon the nitrogenous matters and phosphates of potash and magnesia contained in the wine. Hence it cannot thrive and provoke fermentation in pure alcohol. But he showed that it lived and multiplied in a mixture of alcohol and water, to which, instead of albuminoid matter, he added alkaline and earthy phosphates, with a little phosphate of ammonia, and especially if it were acidulated by a small quantity of acetic acid. This experiment proves that the albuminoid matters in the wine are not, as taught by Liebig and others, the acetic ferment. He revolutionised the mode of making vinegar, averting deterioration and the existence of the eel-like organisms which, prior to his investigations, abounded in every barrel produced according to the Orleans system, thus conferring immense benefits upon a large number of persons employed in its manufacture.

Spontaneous Generation is a Chimera.—It is not difficult to see how these researches led Pasteur to undertake an inquiry, the result of which was to overthrow forever the theory of spontaneous generation. The researches of Pouchet had just excited renewed interest in it. Pasteur had this great advantage over his antagonist. He had been trained in the experimental sciences, whereas the culture of his opponent had been derived from the sciences of observation. When therefore Pasteur asked himself, "Whence do these microscopic organisms come?" he was directly confronted with the problem of spontaneous generation. All Pouchet's statements and experiments were explained and demolished, and the doctrine was wholly discredited, until revived for a short time with apparent

success by our countryman, Dr. Bastian. But the fallacies of Bastian's experiments were exposed and overthrown with characteristic promptitude and precision.

In a celebrated lecture at the Sorbonne, Pasteur, after he had completed his experiments and observations, exclaimed, "There is not one circumstance known at the present day which justifies the assertion that microscopic organisms come into the world without germs, or without parents like themselves. Those who maintain the contrary have been the dupes of illusions and ill-conducted experiments, tainted with errors which they know not how either to perceive or to avoid. Spontaneous generation is a chimera."

Diseases of Wine.—He now reverted to the study of fermentation, as illustrated in the manufacture and the diseases of wine. In the healthy fabrication of red wine the ferment is the *Mycoderma vini.* When this has accomplished its purpose, it withers and disappears, and it is then that, unless precautions are taken, the *Mycoderma aceti* gains the ascendancy, producing acid, sharp, or sour wine. There is another disease favoured by the great heat of summer. The wine is said to turn, to rise, to spurt. It becomes slightly turbid, flat, or piquant. It bubbles when poured into a glass.

Pasteur found that the turbidity was always due to thin filamentous organisms of variable length. The fungus also produces bitterness as the wine ages. The greasiness of wines he found to be due also to a filamentous organism about the 1,000th part of a millimètre in diameter. He therefore arrived at the conclusion that the diseases of red wine were caused by special living ferments, "the germs of which exist in the wine from the moment of the original fermentation which gave it birth."

Having discovered the cause, it became necessary to endeavour to find the remedy. Here remark the quality of the genius brought to bear upon the point. The object aimed at was to devise some method which, whilst harmless to the wine itself, would not alter its bouquet or taste. After many fruitless trials, he hit upon the idea of trying heat. The result was, that by raising the wine for a few seconds, after being bottled, to a temperature of from 55° to 60° Cent. (122° to 140° Fahr.), the organisms were killed, and all ulterior changes of a fermentative character prevented. The process causes no injury to the wine.

The Silkworm Disease.—In 1849, after an exceptionally good harvest had been garnered in, the symptoms of the silkworm disease first made their appearance, and went on

spreading till, in 1865, the malady had almost ruined the silk industry in France. It had also extended to Italy and India, so that about the only silk-producing country which had escaped the epidemic was Japan, whence eggs were procured for cultivation. The widely diffused distress thus occasioned may be realised, when I state that in the South of France the livelihood and life of the main bulk of the population in many districts hangs on the existence of the silkworms. They abound in every house, and tier upon tier are often made in sheds for their accommodation. Great and small silkworm-rearing establishments exist everywhere. When people meet each other, instead of saying, "How are you?" they say, "How are the silkworms?" They get up in the night to feed them, or to keep up around them a suitable temperature. The least change of weather is watched with the utmost anxiety. "Will the mulberry leaves be wet? Will the worms digest well?" For the condition of the food upon which they live, and the maintenance of healthy digestion, are essentially of the first magnitude. They seem to do nothing but eat all their lives! During the last days of rearing their appetites are said to be insatiable. Day and night their attendants stand at attention; sacks of mulberry leaves are incessantly brought in and opened out on litters. In the extensive tiers of specially constructed sheds, the munching of these leaves resembles the noise made by rain falling upon thick bushes. When they arrive at the last moulting, with their bodies swollen with silk, they mount upon the brambles prepared for them, and there shut themselves up in their golden prisons and become chrysalides. The harvest soon follows in gathering in these cocoons, and seldom fails to produce a good return.

This was not so, however, during the progress of the great epidemic, to the cause and cure of which Pasteur was moved to address himself by his friend and former master, M. Dumas. About this time, M. de Quatrefages pointed out to the Academy of Sciences that the Italian naturalists, Filipi and Cornalia, had discovered in the moths and worms vibratory corpuscles, only to be seen by the microscope; that Dr. Osimo, of Padua, had found them in some eggs; and that Dr. Vittadini recommended a microscopical examination of these eggs, with a view to separate the sound from the unsound ones. It was this passing remark, to which little importance seemed to be attached, that probably attracted Pasteur to the study of the silkworm plague. He had already demonstrated, by means of the microscope

and innumerable experiments, how infinitely great were the functions performed by the infinitely little; and he was fascinated by the thought of again resuming a congenial means of research.

Although Pasteur at first declined the suggestion, when once he had been persuaded to undertake the duty, he hesitated no longer. "I attach", said Dumas, "an extreme value to your fixing your attention upon the question which interests my poor country. Its misery is beyond anything you can imagine." "But consider", replied Pasteur, "that I have never handled a silkworm." His old master rejoined: "If you know nothing about the subject, you will have no other ideas than those which come to you from your own observations."

On June 6th, 1865, he started for Alais, the spot where the plague raged with the greatest intensity. Within three hours of his arrival, he demonstrated the presence of the corpuscles in certain worms. To make a long story short, he examined the eggs, the worms, the chrysalis, and the moth. He found that the seed of the disease might exist in the egg and escape attention; that it might exist also in the worm and elude discovery by the microscope; but in the moth the germs were so fully developed as to admit of the readiest recognition. By an elaborate series of observations, he found that healthy moths supplied healthy eggs, healthy eggs produced healthy worms, and healthy worms evolved fine cocoons; and all that was necessary for the restoration of the silk husbandry was the separation of healthy from unhealthy moths—the elimination of the latter, and the exclusive employment of the eggs of the former. This is now done on the largest scale. So simple is the remedy, that the microscopic examination of the moth is entrusted even to boys and girls.

The germ-theory of infectious disease was beautifully illustrated. Worms were infected by feeding on leaves smeared over with corpuscular matter; by inoculation; by each other through the wounds or scratches of their own claws; by simply mixing the healthy with the unhealthy; and by the wafting of corpuscles through the air. The disease was called *pebrine*. Pasteur also pointed out the cause and cure of another serious malady—*flacherie*—affecting silkworms, with complete success.

Study of Beer.—In October 1868 Pasteur was struck down, in the midst of his work, by paralysis. But in a few months we again find him combating the criticisms

to which his inquiries had been subjected with unflinching energy. Moreover, he took up the study of beer, and traced the sourness, putridity, and other diseases of beer to "special ferments", altogether differing in form from the *torula* or yeast-plant. He cleared up many mysteries of our breweries, and explained how much loss was often sustained by the use of bad yeast, containing the germs of other organisms which overpower and destroy the true *torula*.

Anthrax, or Splenic Fever.—Pasteur was at length, by a process of evolution, induced to address himself to the relations of germs, bacilli, bacteria, and bacteridia to the infectious diseases of animals, men, women, and children. All his previous researches and discoveries, after he parted company with the science of molecular physics, led him irresistibly to this point. Fortunately, the infirmity from which he laboured had in no way impaired his zeal, enthusiasm, energy, and mental powers. Not being a physician himself, he hesitated, it is true, to encroach upon a domain of the practitioners of the healing art; but he did not fail to note the interest which had been taken by physicians and surgeons in his experiments and discoveries, and how, in the hands of our illustrious countryman, Sir Joseph Lister, the practice of surgery had been revolutionised and improved by the recognition of the influence of some of the members of the third kingdom in aggravating or causing disease. He therefore associated with himself a physician and former pupil, M. Joubert, who was well versed in the experimental sciences. He thus prepared himself to investigate the cause or "etiology of contagious diseases, which was on the eve of having unexpected light shed upon it". He first applied the principles which for twenty years had directed his labours to the study of anthrax, or splenic fever, and which was supposed to vary with the species that was attacked by it. In the sheep it was called *sang-de-rate;* in the cow, *maladie du sang;* in the horse, *splenic fever;* in man, *malignant pustule, charbon,* or *milzbrand.*

In 1850, Davaine and Rayer had found in the blood of animals which had died from splenic fever microscopic rods or bacilli, but attached no significance to the discovery. In 1863, Davaine's attention was attracted to the researches of Pasteur, and declared his conviction that these bacilli were the cause of the fever. In 1876, Dr. Koch, a young German physician holding a minor appointment at Breslau, following up the life-history of the microbe, proved it to

be a contagium of the fever by a prolonged, clear, and convincing array of experimental observations. He also showed that, whilst the bacillus itself might be killed, the disease could be lighted up by its spores or seeds, and that these were endowed with extraordinary tenacity of life. He further pointed out that, whilst mice and guinea-pigs were always killed by the organism, fowls were not fatally affected by it.

Pasteur was struck by this peculiarity. Why should birds enjoy an immunity from the action of a bacillus, a few of which, introduced into the blood of an ox, will unfailingly prove mortal? "The explanation is", says Tyndall, "a hand-specimen of the genius of Pasteur, which strikingly illustrates its quality." He proved that the microbe of splenic fever dies at a temperature of 44° Cent. (111° Fahr.). The blood of birds stands at 41° and 42°. Does not the vital resistance suffice to bridge over the small gap between 41-42° and 44-45°? Owing to this, the avidity of the blood-globules for oxygen is so great that the bacillus cannot obtain any to live upon. Hence it perishes; and hence the immunity. If so, the cooling of the blood would enable it to live, thrive, and develop.

A fowl was inoculated with the bacillus of splenic fever, and its feet placed in water at 25° (Cent.). Its temperature was reduced to 37° or 38°. In twenty-four hours it died, and its blood was found crowded with splenic fever bacteria. But might it not have been restored by again raising the temperature? In another experiment the cooling treatment was applied after inoculation, and, when the fever was at its height, it was removed, wrapped in cotton-wool, and placed in an oven heated to 35°. It gradually recovered, as the normal warmth of its blood became re-established, and in a few hours was restored to health and vigour; it was killed, but no microbe could be found in its blood.

Fowl-Cholera and Vaccination.—Pasteur next confirmed the dependence of fowl-cholera upon a bacillus of a specific kind. Having done this, his next step will ever be memorable in the annals of medicine. He has proved that septicæmia, splenic fever, and fowl-cholera are caused by living organisms, and he applied this to all infectious disorders. The non-recurrence of attacks of most of these puzzled him. Why should this be so? The question has often been asked, but could not be satisfactorily answered until the germ-theory had been established by the researches of Pasteur. The true answer was doubtless greatly

facilitated by his discoveries of the processes of the various kinds of fermentation, in which he demonstrated that the tartaric acid, alcoholic, acetic acid, and butyric acid ferments were simply living organisms requiring suitable food for their germination, development, and sustenance. So with reference to the bacillus of splenic fever, the vibrio of septicæmia, and the micrococcus of chicken-cholera. They must have a congenial soil on which to feed, live, thrive, and breed.

The particular organism of many of the infectious fevers has not yet been discovered, probably being so minute as to defy detection by the highest magnifying powers available. Pasteur believes that in all human probability, when they have been isolated, they will be found to be forms of micrococci. These fevers all comport themselves like those in which microbes have been proved to exist. Thus, there is a period of incubation or hatching, a certain lapse of time between the introduction of the organism into the system and the symptoms to which it presumably gives rise; a definite course; infectiousness, and non-recurrence in most cases.

When the organism has used up the material on which it lives and multiplies, it perishes from want, the symptoms subside; and, as it takes a long time before the soil is renewed, a course of such parasitic cultivation in the system is rare. The protection thus afforded often lasts a lifetime. Here, in a few words, we have the whole explanation of the protective power of vaccination.

Pasteur recognised the vaccine virus as a weakened form of the small-pox virus, so attenuated by having first passed through the system of the cow, but vigorous enough to exhaust the soil on which the variolous microbe would otherwise graze and increase, and that without serious risk being incurred of a fatal result. If, he probably reasoned, protection can be given for a considerable period against small-pox by vaccination with a benign virus, why cannot the same principle be applied to all other non-recurring contagious diseases?

No sooner was this conception formed by Pasteur than he, with characteristic ingenuity and energy, undertook a series of experiments with a view to attenuate the virus of several infectious diseases whose microbes had been fully investigated. He first began with chicken-cholera. He made a great number of successive cultivations of its micrococci in quick succession; but he found it was as fatal at the end as at the beginning. It had not been left,

at each cultivation, sufficiently long under the influence of oxygen. He now left each cultivation a long time exposed to the air; and he was at length gratified to find that by this process he could at will produce a chicken-cholera vaccine, which, when used, caused fowls to sicken only and not to die. Moreover, when, after the vaccine indisposition has passed away, inoculation with the most virulent virus produced no effect whatever. The protection was complete. The vaccination with the benign virus had so exhausted the soil that nothing remained for the micrococcus to feed on and multiply.

Vaccination as a Prophylactic of Splenic Fever.—This discovery led Pasteur to try to attenuate the virulence of the splenic fever microbe so as to convert it into a true vaccine. He succeeded, after prolonged inquiry and experimentation. The following test-experiment will show the extreme value of the discovery. On the 5th of May 1881, at Melun, twenty-four sheep, one goat, and six cows were each inoculated—or, as we ought to say, vaccinated—with five drops of attenuated splenic fever virus. On the 17th *idem*, these thirty-one animals were revaccinated with a slightly stronger virus. Twenty-five sheep and four cows were left unvaccinated. On the 31st of May all the vaccinated and unvaccinated animals were inoculated with virulent virus. On the 2nd June more than 200 persons met to witness the result, including Prefect Patinot, senators, general councillors, journalists, a great number of doctors, veterinary surgeons, and farmers; but, of the twenty-five sheep which had not been vaccinated, twenty-one were dead (the goat was also dead); two other sheep were dying: and the last, already smitten, was certain to die that very evening. The non-vaccinated cows had all voluminous tumours at the point of inoculation, behind the shoulder. The fever was intense, and they had no longer strength to eat. The vaccinated sheep were in full health and gaiety. The vaccinated cows showed no tumours; they had not even suffered from an elevated temperature, and they continued to eat quietly. The sceptics—and they were many—were amazed and silenced. M. Rouley said to them: "There is nothing for you to do but to bow before the master," pointing to Pasteur, "and to exclaim—'I see, I know, I believe, I am undeceived!'"

The practice has been widely adopted in France. The mortality (1881) in sheep has been ten times less, and in cows fourteen times less, from splenic fever; and in 1882 the success of the system has been well maintained, all due

to vaccination with attenuated virus. At the end of 1881, nearly 34,000 animals had been vaccinated; in 1882, almost 400,000; and 1883, close upon 500,000.

Vaccination as a Prophylactic of Hydrophobia.—Pasteur will be seventy-one years of age on the 27th of December. In spite of a life of incessant labour and combat, and of his having been a constant sufferer from the effects of a stroke of paralysis or hemiplegia during the past twenty-three years, he has never faltered nor swerved from the energetic pursuit of the grand purpose of his life—the study of the habits and the life-history of what Pouchet loved to call the invisible world, or the infinitely little, or the third kingdom of Dumas.

In 1880 he was introduced to his first case of hydrophobia. He at once recognised it as a malady probably of microbic origin. But, so far as I know, the organism has not yet been isolated and subjected to pure cultivation like those of septicæmia, splenic fever, and chicken-cholera. Although the cause or virus exists in the saliva of the rabid animal, and in that of man, he found it, in its purest form, in the brain and spinal cord, also in the trunks of the nerves. He has subjected it to attenuation under the influence of oxygen, and succeeded in so far enfeebling it that he now employs it as a safe vaccine for the prevention of rabies and hydrophobia.

His discovery was reported upon in May 1886 by a Commission. "It inspires", says Tyndall, "the sure and certain hope that, as regards the attenuation of rabic virus, and the rendering of an animal by inoculation proof against attack, the success of M. Pasteur is assured." The results then obtained were: 1. Of six unvaccinated dogs bitten by a rabid dog, three died from rabies. 2. Of eight unvaccinated dogs six died rabid after the pure virus of rabies had been injected into a vein. 3. Of five unvaccinated dogs, all succumbed to inoculation of the brain with rabic matter. 4. Of twenty-three dogs vaccinated with attenuated virus, and subsequently inoculated with the strongest virus, not one was attacked with the disease. The system has been favourably reported upon by an English Commission.

Pasteur and his coadjutors are satisfied with the success which has attended the practice of the system during the past five years. There has been, and is still, opposition. So it was with his discoveries in molecular physics fermentation, the silkworm disease, and spontaneous generation. In like manner, the germ theory of disease, so beautifully illustrated by his investigation and treatment

of the silkworm epidemic, by the researches of Budd in medicine and Lister in surgery, has been tardily accepted. We need not, therefore, be surprised at the scepticism which prevails about the efficacy of the rabic or hydrophobic vaccine. Experience, not only in reference to this, but in most other cases, shows that it is unavoidable, and to be expected. Science is uncompromising and exacting; and it is well that she should be. This places the explorer upon his mettle; it operates as a wholesome stimulant, and tends to improve the quality of the work done.

No scientist in this or any other age has accordingly fought so many battles as Louis Pasteur; and none, living or dead, has won so many glorious victories. He has never, to my knowledge, suffered a serious reverse. In the language of Tyndall, "A great scientific theory has never been accepted without opposition. The theory of gravitation, the theory of undulation, the theory of evolution, the dynamical theory of heat, all had to push their way through conflict to victory." Every discovery by this great man has been but the prelude to others, and the last to which I have referred is the normal sequence of those that have preceded it. His life-work is one grand evolution. Medicine and surgery have been immensely advanced by the assimilation of the principles which he has established, and how much more the improvement may extend may be divined, not measured just yet, by the extraordinary development which has taken place in bacteriology during the past twenty years.

During the greater part of this period, bacteriology has been assiduously cultivated by many eminent scientists in many lands. The result is a vast accumulation of knowledge regarding the causation of zymotic affections. The outcome up to date was concentrated in the Bacteriological Museum of the Congress of Hygiene and Demography lately held in London. But it was apparent, to an onlooker like myself, both from the numerous specimens presented and the discussions in the section of bacteriology, that, valuable as most of the investigations have been, it must be admitted that there is room for correction and revision. Not long ago, the microbe, or micrococcus, as the case might be, was considered sufficient to cause definite and specific morbid phenomena. Now we find a tendency in some instances to ascribe these to their products. How far this may be so, time and experience will show. It is well, however, to bear in mind that such common phenomena as

scabies, many cutaneous and other affections, are directly due to living things, and not merely to their secretions.

In all times and climes the unbalanced relation of growth and decay, especially the abnormal preponderance of dead organic matter, under conditions favouring decomposition and putrefaction, has always been fraught with the proportionate prevalence and dissemination of infectious disease. On the other hand, the restoration of a natural balance between vitality and death, or the excess of living over dead organic matter, has been marked by a tendency to the diminution and prevention of such disorders. The reasonable explanation of this is, that the nidus of dirt and filth, in various forms and degrees, in which pathological organisms germinate, develop, grow, and propagate, being destroyed by the extension of hygiene and sanitation, the microbes causing specific diseases are deprived of congenial sustenance and starved. Hence the vast importance of "the cleanliness which is next to godliness"; purity of air, earth, water, food, and the person; the best possible clothing and housing. How much has been done, how much remains to be accomplished, is demonstrated by the subjoined tabular statement compiled from the Fifty-second Annual Report of the Registrar-General, p. 45 (1889):—

Annual Death-Rates from the Principal Zymotic Diseases to a million per thousand living in England. In Groups of Years, 1858-89.

Cause of Death.	3 Years. 1858-60.	5 Years. 1861-65.	5 Years. 1866-70.	5 Years. 1871-75.	5 Years. 1876-80.	5 Years. 1881-85.	4 Years. 1886-89.
All causes	22221.3	22582.0	22424.6	21962.4	20791.0	19298.6	18433.0
Small-pox	219.3	218.6	104.8	410.8	78.4	78.0	16.3
Measles	480.0	456.6	428.4	373.2	384.8	410.0	468.5
Scarlet Fever ...	891.0	982.4	959.8	758.6	679.6	434.0	236.5
Typhus	792.0	921.8	849.8	81.4	34.2	22.8	6.8
Enteric Fever				373.8	277.2	215.0	176.5
Simple and Ill-defined Fever				140.2	69.2	34.0	17.3
Whooping-Cough ...	494.3	515.8	545.0	498.6	527.0	456.6	427.7
Diphtheria	372.3	247 6	126.8	120.8	121.8	155.6	164.2
Cholera	32.0	42.4	172.4	30.6	20.4	16.2	13.5
Diarrhœa, Dysentery	777.3	874.0	1062.8	1000.4	832.4	652.4	671.7
	4058.2	4259.2	4249.8	3808.4	3025.0	2474.8	2199.5

That whilst material advance has been made in this country in the reduction of the death-toll from small-pox, scarlet fever, typhus fever, enteric fever, simple and ill-

defined fever, diphtheria, and cholera, it remains stationary, or nearly so, from measles, whooping-cough, and it is not much affected in diarrhœa and dysentery. Changing the ratio of mortality in each group from per million to per thousand, there is presented the extent of the decrement in the commonly used formula that has been effected in recent times. Thus, in three years, 1858-60, the death-rate was 4.0582; in five years, 1861-65, 4.2592; in five years, 1866-70, 4.2498; in five years, 1871-75, 3.8084; in five years, 1876-80, 3.0250; in five years, 1881-85, 2.4748; in four years, 1886-89, 2.1995. As all the ten infectious diseases enumerated in the first statement are admittedly preventable, the result displayed by these figures is not really so satisfactory as it should have been. "Then", in the weighty words of the Prince of Wales, expressed in his opening address to the Congress of Hygiene and Demography in August last, "why are they not prevented?" A part of the reason was given by the President, Sir Douglas Galton, in his speech closing the great gathering, when he remarked that it was to be found in imperfect education (in sanitary matters) of the municipal electorate and their representatives. The other moiety of the reasons is discoverable in the existence of a property electorate in London especially, which gives the owners of insanitary houses and areas an undue preponderance of power in maintaining things as they are; in charging the ratepayers an exorbitant price for houses condemned as unfit for human habitation; in the Legislature not having as yet simplified and cheapened the complex legal forms of purchase, and tapped considerable sources of wealth which now escape taxation, as indicated in another portion of this address, the proceeds of which might be devoted to the demolition and renovation of unhealthy premises and areas.

Epidemics.—Infectious diseases in an epidemic form have prevailed at times from the remotest periods of the historic record, if not, indeed, in prehistoric times. Armies in the field and in quarters have often been decimated, nay, virtually destroyed by them; sections of the human race have been removed by their rapid march and diffusion, whilst the intellectual life and advanced civilisation have been checked or stopped by them. The power and civilisation of Greece and Rome were materially affected by the deadly epidemics which prevailed between "antiquity and the Middle Ages". About the middle of the fourteenth century the Black Death not only diverted the course of

"intellectual and social activity", but it stamped its degenerating and blighting impress upon "the developments of succeeding centuries". In Basle, fourteen thousand; in Venice, three-fourths of the people; in Germany, a million; in Italy, a half of the inhabitants; and in England, nine-tenths of the population, are reported to have perished from this pestilence. In ancient times, the sweating sickness and the plague created widespread havoc in Europe, Asia, and Northern Africa. Remains of bubonic plague have appeared, in modern times, at Pali and Kumaon in British India, and more recently in some parts of Arabia, North Africa, and in Russia.

As the ordinary explanation of the origin and diffusion of infectious diseases did not appear to satisfactorily meet all their conditions in an epidemic form, our ancestors had recourse to various agencies as lying at the foundation of their causation and spread. Thus their extraordinary extension was attributed to a *constitutio pestilens*, a *genius epidemicus*, an epidemic constitution of the atmosphere, and more recently to epidemic influence, pandemic waves, climatic or climatical agency, electrical, magnetic, telluric and kosmic influence, or to excess or deficiency of ozone. These epidemics were frequently associated with some mysterious power of the sun, moon, planets, and their constellations, stars, comets, earthquakes, volcanic eruptions, inundations, "spasmodic convulsions of the sickened earth", or with "a peculiar corruption of the air, which was often expressly declared to be thoroughly immaterial and dynamic".

Although the germ theory as expounded by Pasteur, Klein, Lister, Koch, Burdon Sanderson, Cruikshank, and a host of other bacteriologists, goes far enough to explain the causation and course of infectious diseases in their endemic forms, it does not, to my mind, altogether account for their rapid spread as epidemics. Something, be it what it may, is apparently superadded to the organism which is the essential cause, and the individual susceptible to invasion. On this point, however, I prefer to retain an open mind.

FAMINE: ITS EFFECTS AND RELIEF.

By Surgeon-General Sir WILLIAM MOORE, K.C.I.E., Q.H.P.

(*Read:* 16*th December* 1891.)

THERE is no lengthened period in the history of the world during which famines have not occurred. When a Pharaoh ruled Egypt, and after Joseph interpreted his dreams, "famine was over all the face of the earth." The old empires of the Euphrates, Assyrian and Babylonian, were not free from the scourge. Imperial Rome itself suffered from famine. Parts of Europe in the Middle Ages were frequently famine-stricken. At more recent dates, China, especially in the north, has experienced famines. Famines in India have been familiar to living men; especially in 1865, in 1868-69, in 1874, and 1877. Even the United Kingdom has not been free from famine during the present century, as the Irish famine of 1845-46 testifies. Now we have distressing accounts of the famine in Russia and in India. Mr. Cornelius Walford, in the *Statistical Journal*, vol. xli, mentions no less than 350 historical famines.

Famines, like pestilences, have been attributed to many causes. In tropical and semi-tropical countries the most frequent cause is undoubtedly drought, although this does not agree with what Mr. F. C. Danvers states in his *Report on Famines in India.* Mr. Danvers says: "The most general cause has not been failure of the rains."

But all the famines and scarcity of food I have known in India certainly originated in failure of the rains. Another cause of famine in such almost rainless countries as Egypt, may be failure of the usual rise of the river, which imparts fertility to the land. Even now schemes are under consideration for the extensive storing of Nile water to provide against failure of the annual overflow: schemes which we may safely anticipate will come to naught if Mr. Gladstone returns to power and scuttles us out of Egypt. And here I remark that the importance of storing Nile water has been recently emphasised by Mr. Cope Whitehouse, who points out that Emin Pasha can now control the Equatorial Nile. Mr. Whitehouse observes:

"Before Emin Pasha is declared an outlaw, his means of offence as well as defence must be considered. He can next summer inflict a fine of £10,000,000 upon Egypt by simply throwing a temporary embankment across any important affluent, ... which would compel Europe to treat with him on his own terms or retake the Soudan before the low Nile of 1893."*

Heavy or long-continued rains, especially in a tropical country, may also be a cause of famine, the seeds or shoots rotting in the ground from too much moisture. Inundations from rivers or the sea have also caused famines by destroying the crops over large tracts of country. The Orissa famine of 1856-66 was caused partly by drought and partly by inundation. Hurricanes, hail-storms, earthquakes, severe frost, have all been more or less causes of famine. Rats eating the crops have caused famine, as, for instance, in Madras in 1812. Insects may also destroy crops to such an extent as to cause famine. Scarcity frequently, and famine often, have been caused by locusts. Locusts were among the plagues of Egypt. It is written: "Before them were no such locusts as they, neither after them shall there be such." I think, however, that the locusts of modern times are almost up to the record. In articles on locusts published in the *Indian Medical Gazette*, 1872, I thus described the locusts which were the cause of the second year of famine, 1869, in Rajpootana: "The people (estimated at some 2,000,000) who thus left Rajasthan on the failure of the rains of 1868, remained subsisting as best they could until the rains of 1869 commenced. They then streamed back to their villages, and, hopeful for the future, prepared the ground, and put in the seed which had been saved. The rains were favourable, the crops sprouted, and the country once more assumed a prosperous aspect. But when the crops were less than half-grown a new calamity befell these districts. Locusts numerous as the sands of the sea appeared, and in a few days not a green thing was left. . . . The numbers of the locusts cannot be imagined. . . . For miles my horse crushed them at every step." General Brooke, the historian of the Rajpootana famine, described how they invaded the towns and villages, driving the people from their dwellings. Although locusts are so destructive, they are preyed upon by various kinds of insects, birds, and quadrupeds. In some countries the inhabitants eat locusts, but this is not common in India, except during famines. In Africa,

* *Morning Post*, November 21st, 1891.

Livingstone said, they were a blessing to the people wanting food. Unless disguised by condiments, locusts, when feeding on vegetation growing in a saline soil, are in flavour not unlike shrimps. Parke, in his experience in Equatorial Africa, writes of occasional meals of locusts, which, "when placed on a pan, give one hop, a crack, and are cooked." *En passant* it may be observed that the ancients employed locusts in medicine. Dioscorides said that the thighs of locusts, singed with goats' blood, is a cure for leprosy.

Defective transport is also a cause of, or at least aggravates, famine. In India it has occurred that while there was plenty in one district, famine prevailed in an adjoining country. Because the famine was what the Indians term a "teen-khal", or a threefold famine. There was neither grass, grain, nor water. Therefore beasts of burden could not be used to import grain into the famine-stricken districts. This was sorely felt in the Rajpootana famine before referred to. From Deesa in the south to the Punjaub in the north, a distance of some 400 miles, and from Scinde in the west to Central India in the east, a distance of 450 miles, there was no railway communication. The poor people, therefore, from an area of nearly 7,000 square miles, were obliged to emigrate in search of food for themselves and their cattle, as food could not be taken to them. This they did in four great streams. One division went north to the Punjaub; one division went south to Guzerat; one division, the smallest, went west towards the Indus; and one division, the largest, went east over the Aravelli Mountains, through the comparatively less famine-stricken principality of Meywar into Malwa and Central India. From Marwar alone one million people emigrated, taking with them two millions of cattle. It was estimated that during the two years of famine (the first caused by failure of the rains, the second by locusts) one million and a quarter died, either purely from famine, or from diseases caused by famine.

Here I wish to remark that it has been asserted by anti-opiumists that the cultivation of opium in India is so extensive that sufficient land does not remain for the growth of cereals, and thus famine results; the fact being that it is only a decimal portion of the cultivatable land of India on which opium is grown. But the most astounding statement which has been made in this connection is, that "the cultivation of opium in Malwa diminished the agricultural products to such an extent, that in the neighbouring province of Rajpootana 1,200,000 human beings

died from starvation, or the diseases caused thereby"! The fact being that hundreds of thousands of human beings found food in the opium-growing country of Malwa —a country which was to these emigrants, notwithstanding its opium cultivation, a land overflowing with milk and honey in comparison with Marwar or Bickaneer, with their sterile plains, Shekawutty and its sand-hills, or even Meywar, with its lakes dried up. It has also been insinuated that the Orissa famine of 1865, during which parents are said to have eaten their children, was caused by opium cultivation. The anti-opiumists, like the anti-vaccinationists, although unable to control the logic of facts and figures, are an active and noisy body, and have many adherents, to whom statistics are a maze and common-sense a myth; so they are ready to believe anything. Now there is no more connection between famines and opium-growing in India than between German literature and a German flute. The Orissa famine was caused by the failure of the rains of 1865, extended by the inundation of 1866, when 1,049 square miles were flooded, and aggravated by the want of roads throughout the country. After the Orissa famine an Act was passed providing for more roads, and local boards were appointed on the lines of British Road Boards, reminding somewhat of locking the stable-door after the steed was stolen. The Rajpootana famine was caused first by the failure of the rains of 1868, extended by the ravages of locusts in 1869, and aggravated by the absence of a railway, since constructed. Instead of opium being a cause of famine, it was the salvation of thousands. It enabled them to bear up against hardships to which, without opium, they must have succumbed. To a certain extent it supplied the place of food. It in some degree delayed that bowel-complaint which is so usually met with in the famine-stricken; and it exerted a certain prophylactic influence over the fevers which prevailed during, but especially after, the famines.

Famines have been attributed to comets, sun-spots, etc.; so have many diseases, especially cholera. Old superstitions, like old religions, take a deal of killing. It has not been proved that famines are more prevalent when comets or sun-spots have been noticed; and, notwithstanding what has been advanced about sun-spots by Messrs. Lockyer and Hunter,* the connection with famine cannot be upheld. Lastly, deadly war hath his coine of

* *Nineteenth Century Review*, 1877.

pestilence and of famine. Famine also hath its sequence of disease.

Perhaps there is no better description of the famine-stricken than that given by Ovid. "There she found Famine in a stony field, scratching up a few roots with her talons and teeth. Her locks were matted; her eyes were sunken; paleness overspread her face; her lips were wan; her teeth brown with rust; her skin was hard, and through it the entrails were seen to move; the sapless bones seemed to start from her bent loins, and for a belly was a belly's space. Thou wouldst have supposed that her breast was hung up, and tacked to her body, only by the chine of her back. Her joints were protuberant from leanness. The orbits of her knees bunched out, while her ankle-bones jutted to undue proportions."

I have had too many opportunities of seeing the famine-stricken in all stages in Kutch, Bhooj, Rajpootana, and the Presidency of Bombay. I marched through the famine districts of Rajpootana for a thousand miles. I have seen the people digging for roots as Ovid describes; I have seen them emaciated to skin and bone; I have seen them dying by the road-side or in the jungle, the vulture and the pariah in close attendance. I have seen their corpses by the roadside or in the jungle, partially devoured by wild beasts or by birds of prey. This was chiefly in the Native States, where, as previously observed, there being neither grass, grain, nor water, nor a railway, food could not be taken to the people. I have known them come and leave their children on the premises of British officers, and then stagger into the jungles to die. And here I may mention that among the various officers who afforded relief to the Rajpootana famine-stricken the services of Colonel Carnell (now Major-General), commanding the Erinpoorah irregular force, were conspicuous and acknowledged by Government in the following terms: "In command of the isolated out-post of Erinpoorah, through which the returning tide of famished emigrants streamed in countless thousands, you devoted all your energies to the relief of the great distress. . . . . Subsequently in charge of the special relief-house for the children rescued from famine . . . . for all which the Government of India desire to express their warm gratitude." General Carnell sent to the surrounding villages, and collected nearly 1,000 starving children, many of whom were afterwards claimed. All honour is also due to the Rev. Mr. Schulbred, of the Presbyterian Mission, who collected some 300 children, housed, fed, and educated them,

eventually locating them in Christian villages. It was not until afterwards, when famine appeared in British Provinces, that other rewards than thanks were given to officers for conspicuous services during famine. But such work as was done by General Carnell and Mr. Schulbred should not be forgotten. Chaucer of old said, "au shulde hire children sterven for famine." But these gentlemen did better still.

Here I mention that during famine periods I noticed that neither pariah dogs nor birds of prey looked happy or healthy. A great mortality occurred among the dogs, and the melancholy condition of other carrion feeders appeared to result from repeated surfeits of human and animal flesh.

It may be truthfully stated that more people die from the diseases arising directly or indirectly from famine than from actual starvation. It is not given to all men to be Succis or Tanners, and so to fast without detriment. Mr. John Griscom, of New York, certainly declares that fasting is all a matter of faith, and that any person who will begin a fast with the full belief that it can be done without harm will succeed; yet, notwithstanding this, we all know that, whether from absence of faith or from absence of food, anæmia is developed by want, or by poor or insufficient diet, not amounting to actual starvation. When famine has occurred in India, large numbers of the population, whose circumstances placed them above actual want, have become anæmic. As want becomes more pressing, the person grows more exhausted, and probably suffers from palpitation, giddiness, and from constipation. There is loss of weight, subcutaneous fat disappears, and the muscles waste. The skin of the face becomes tightly drawn over the protruding features, and that of the body baggy. There is also often gastro-intestinal irritation, and generally one of the specific maladies afterwards mentioned. It has been stated that the skin frequently becomes covered with a brown, filthy-looking coating, and that the body emits a fœtid odour. The brown coating I regard as dirt, and the filthy odour as its result.

In my experience, accompanying the first physical results of starvation there is mental weakness. The mental condition soon evidences that the brain participates in the mal-nourishment. What contributes much to the mental lethargy is grief and sorrow. The cultivator daily and hourly watches anxiously for the clouds which do not come to

dim the sky of brass, and to discharge moisture on the iron earth. He applies to the local rainmaker, whose incantations are in vain. He sees his crops fail, and his scanty means of sustenance being rapidly exhausted. He hears his family crying for food which he cannot give. He sees his belongings become haggard, exhausted, emaciated, and diseased. And he sees his cattle die. For misfortune comes not in single file, but in battalions. Thus *atra cura* is always with him. The apathy, the result of starvation, has been well illustrated in all the famines I have witnessed. Many although offered what might be considered light employment, and good food or pay, would not work. Indians, probably by reason of generations living under a tropical sun, are naturally more or less apathetic. The belief in *Nusseeb* or fate also exerts a potent sway. "What has been has been; what will be will be," dominates the mind during ordinary circumstances, and more especially so when the brain is enfeebled by mal-nourishment. Sympathy for the famine-stricken has often been diminished by the disinclination to work evinced. But this disinclination is the result of starvation on peculiar temperament, as far as Easterns are concerned, and the people should no more be blamed for it than for any other disease starvation causes. One of the usual results of famine is pain and irritability of the stomach, with first constipation and then the reverse, diarrhœa or dysentery. The investigations of Dr. Cunningham during Indian famines have shown that the process of chronic starvation is accompanied by a shrunken and contracted condition of the stomach and intestines, and an atrophied state of the viscera. Also by desquamation of epithelium, and changes in the intestinal structure of a fatty nature, which must render them more or less unfitted for their functions.

It may, however, be questioned if this is the only cause, or even the general cause, or the primary cause, of famine bowel-complaint. In the first place, the natives of India and of other tropical countries are especially liable to bowel-complaints. Among the rice-eating population of Bengal, diarrhœa is so common, that a former President of this Society, the late lamented Chevers, termed it *morbus Bengalensis;* and he wrote: "Born of a poorly-fed race, nursed by a semi-starved mother, working hard on food the use of which is semi-starvation, want of nutrition causes diarrhœa to present from slight atmospheric changes." In addition to this predisposition, there is the nervous or mental anxiety previously referred to. In any climate,

and at any time, both an acute and a chronic diarrhœa may be excited or maintained by worry or anxiety. There is also another potent factor. The bowel-complaints of famine are as often as not caused by the articles which the people use as food when cereals are scarce or not to be procured. And it may be that these articles tend to induce that condition of bowel which has been attributed to starvation, as they certainly cause diarrhœa of a lienteric nature. Such articles were investigated by Dr. George King and myself, and described as "The Famine-foods of Marwar." They are thus enumerated in my annual "Rajpootana Dispensary, Vaccination, Jail, and Sanitary Report", published as No. cviii, Selection from the Records of the Government of India in the Foreign Department.

1. *Kejra*, the bark of the *Acacia leucophlœa*, a stunted tree growing in considerable abundance on the sandy plains of Western India. 2. The pods of the same tree. 3. *Bharout*, the seeds of the *Achyranthes aspera*, a species of grass, with the spikes of which the traveller soon becomes unpleasantly acquainted after entering the semi-desert districts. 4. *Bhoo-burya* or *mukkia*, also a species of grass. 5. *Massia* or *maleecha*, the seeds of a grass. 6. *Mutteree* or *turbooz-ka-tej*, the seeds of a cucurbitaceous plant. 7. *Nagurmoth*, the root of a tall rush of the order *Cyperaceæ*, which grows on the moist beds of tanks. 8. *Gokhur kantee*, the capsules of the *Tubulus lamegenosus*. 9. *Tilli*, the refuse of the *Sesamum Orientale* remaining after oil has been expressed. 10. *Singara*, or water-chestnut, *Trapa bispinosa*. 11. Lizards. 12. Locusts.

The famine-foods used in the Deccan were also investigated by Brigade-Surgeons Gray and Lyon of Bombay. The principal are as follows: *Abutilon muticum* seeds; the fruit of the *tribulus terrestris;* the seeds of *indigofera limfolia;* the seeds of *indigofera cordifolia;* the seeds of *indigofera glandulosa;* the seeds of *sesbania Ægyptiaca;* the pods of *alysicarpus rugosus;* the seeds of *phaseolus tribulus;* all except the two first being leguminous plants. The seeds of *cyanotis axillaris*. All these were submitted to analysis by Dr. Lyon, the chemical analyser to Government; the result showing that the materials obtained from leguminous plants are more valuable and nutritious than was previously supposed. Dr. Shortt investigated the famine-foods used in Madras. These are mainly the fruit of the prickly pear (*cactus Indica*), of the wild fig, of the wild custard-apple, and the leaf of the *sethna Indica*, a plant of the same natural order as the *coco*. Also the pith of the

flowering stalk of the American aloe. This is an introduced plant, and has been used largely for railway fencing. During the famine every stalk along the railway was cut down. When eaten raw it is said to cause bowel-complaint. In Russia, at the present time, the people are using the boiled leaves of the potato, boiled grass, and the refuse of beetroot after sugar has been made.

It will be evident that a diet composed, or partially composed, of the bark of trees, the pods of leguminous plants, wild fruits, especially the wild custard-apple, the prickly pear, the seeds of grasses and melons, the stalks and roots of rushes, the water-chestnut, the refuse of oil- and sugar-mills, boiled grass, locusts, etc., is quite sufficient to excite diarrhœa in a healthy individual, and more than sufficient to excite it in a person enfeebled by want.

All these things are used to a greater or less extent, either separately or together, and if the *res angustæ* does not forbid, they are luxuriously compounded with an allowance of bajree, wheat, or even rice. Even then most of the necessary constituents of food are more or less absent— albuminates, carbohydrates, fats and salts—and sufficient water to drink is not procurable. It has been stated that cannibalism was had recourse to in Orissa. I have endeavoured to trace this report, and am very reluctantly compelled to conclude that the report was disseminated by the anti-opiumists, who, as formerly mentioned, endeavoured to make it understood that famine in India resulted from so much land being taken up for opium cultivation. The picture of parents eating their children was calculated to add to the horrors of famine, and therefore to the awful responsibility of cultivating opium instead of grain.

I do not believe that cannibalism was practised in Indian famines. But I once saw a number of people surrounding a dead elephant, from which pieces of flesh were being hacked by a man who stood up to his middle in the elephant's open body. Pieces of flesh were caught and devoured, in some instances without any cooking; and this by people who ordinarily or wholly lived on vegetable food. Then comes, particularly after the next rains succeeding a famine, when the cold weather sets in, an enormously increased liability to fever. The connection between famine and fever has long been noted. Shakespeare wrote, "Here let them lie, till famine and the ague eat them up." As regards tropical countries, the explanation of the connection between famine and fever is, I think, sufficiently

easy. If chill is not the primary cause of fever, it is admitted by all that chill is quite sufficient to re-excite a febrile paroxysm. There are very few inhabitants of a tropical country who have not, at some time or other, suffered from aguish fever. When, as at the commencement of the cold weather, the loss of heat from the surface of the body is rapid, the effects resulting from deficient supply of food required to maintain the normal temperature of the body are more quickly felt than when the loss of heat from the surface is slow. Hence, in India at least, it is not during, but after the famine, provided it does not extend over two seasons, that most fever prevails. This was well exemplified in the Rajpootana famine. During the first year of famine, when there was no rain, and therefore the setting in of the cold weather was less marked, the proportion of fever-cases treated at all the hospitals and dispensaries in the Province was only 9.3 per cent. to total treated. In the second year, when there were good rains, and famine caused by locusts, the proportion was 75 per cent. The figures could not be much affected by the emigration of the people, for in both years the masses had returned before the feverish season commenced. They would, however, be affected by the death of so many of the population. But had all the people lived, the number of fever-cases must have been much increased.

The type of fever seen during or after famines is not always the same. The type during the Irish famine of 1846 was typhus. The Irish physicians of the day asserted that this Irish fever was particularly and distinctly due to famine. But as a matter of history, the fever in Ireland began in 1842, before the famine. In Germany the type of fever accompanying famine is stated to have been relapsing, and was significantly called "hunger pest". In the Rajpootana famine of 1868-69 the fever was of the so-called malarious type. This was made the subject of special investigation, and all the medical officers then serving in Rajpootana reported that they saw no relapsing fever. So did the officials of the subordinate medical department in independent charge of dispensaries, to each of whom I sent a concise description of relapsing fever. In the Bombay famine of 1876-77 the fever was diagnosed as relapsing, chiefly in consequence of the *spirillum Obermeieri* having been found in the blood of some of the sufferers. But this spirillum has been found in the blood of others not exhibiting any signs of relapsing

fever. It would seem that relapsing fever is not caused by famine. Various famines have occurred without such attendant. If we are to believe that relapsing fever depends on the spirillum, it must be an infectious fever. When relapsing fever prevailed in London in 1870, it was supposed to have been imported from Germany. Hunger does not infect. The subject is important. Because during the Bombay famine it was argued that the spirillum signified relapsing fever, and that relapsing fever signified that the food given to the people on relief works, etc., was insufficient. The spirillum fever of Bombay in 1877 was by some regarded as identical with the relapsing fever of Europe, and as something new in India. But the relapsing type of fever has frequently been noted in India previously, by Amott in 1844, by Lyell in 1852, by Smith in 1868, by Chaud Dutt in 1866, by Garden and others. But, as a matter of fact, any tropical fever may, and often does, assume a relapsing type. So much so is this the case, that Dr. Lyons, of the Bengal service, wrote a book, endeavouring to prove that all fevers are relapsing. In Russia at the present time typhus is reported. It appears to me that, to produce different types of fever, different influences must be in operation. Thus, when there is overcrowding, as there is during famines in a cold climate, the typhus type will prevail. But in India, where the people, especially in a famine camp, live almost *sub Jove*, there will be no typhus. There will, however, be the malarious type of fever, whether caused by the *bacillus malariæ* or by vicissitudes of temperature. And it may be readily understood that if relapsing fever is caused by the spirillum, seasons of activity of such spirillum may be concomitant with a famine. For fever and famine have a common basis in atmospheric and telluric influences which are unfavourable to the growth of crops, but perhaps favourable to the growth of infective parasites. And the more enfeebled the human organism is, the more is it predisposed to become affected by atmospheric influences and by infective parasites.

Another effect of famine is in the liver, the cells of which lose all fat and the organ becomes smaller. This does not, however, appear to induce hepatic diseases, as I have noticed a decline of such maladies during famine.

Scurvy is a malady which generally accompanies famine. This was specially noted in Sweden in 1784; in Russia in 1785; in Hungary in 1803; in Southern Russia in 1823; in Ireland in 1846-47. Among the natives of India, and

especially in some parts of India, there is a latent scorbutic taint extensively prevalent. This may be readily developed into an active condition. The number of persons suffering from scorbutic symptoms during the Rajpootana famine was enormous, and this among the classes who were not actual victims of starvation. Surgeon-General Cornish, C.I.E., in his account of the Madras famine, states, "The gums became swollen and ulcerated, and there was great tendency to ulceration and sloughing on the receipt of small injuries."

Yet many of the people were perhaps consuming more fresh vegetable matter in the forms which have been enumerated as famine-foods than at any previous period. It is, however, the absence of certain constituents of the food, rather than the presence of noxious material, from which scurvy arises; and experience shows that an insufficient or artificial diet, if long persevered in, will produce symptoms of the disease; unless the insufficiency is so great as to produce starvation and fever first.

The, as I regard it, allied disease, or form of scurvy known as *beri-beri*, was frequently seen in the œdematous form during famines. I say allied disease with some fear and trembling, because in the *British Medical Journal*, Dec. 5th, it is remarked, "that observers, especially in India, seem unable to shake themselves free from certain traditions about this disease." Now, I have seen a great deal of *beri-beri*, and I have elsewhere given my reasons for regarding it as a phase of scurvy. Others, however, have gone in for a *beri-beri* microbe, but have failed to explain why the microbe comes only on, at least chiefly, when certain environments prevail.

General dropsy may be the first symptom. During the Madras famine of 1877-78, Surgeon-Major Porter gave an account in the *Ind. Med. Gaz.* of general anasarca with effusion into serous cavities as very prevalent among the famine-stricken. But in one epidemic which came under my cognizance in Guzerat, during the Bombay famine, the dropsical effusion was more or less limited, and in many cases altogether limited, to the abdomen, and numbers were tapped by Brigade-Surgeon Joynt. Epidemics of *beri-beri*, when occurring in jails, have been attributed to confinement, the depression and anxiety arising from it, hard labour, and food just sufficient on scientific principles to support life, acting probably on systems already debilitated by a malarious, scorbutic, or syphilitic taint, or by several of such taints. Very similar remarks apply to a famine-stricken population.

Cholera, in India at least, frequently accompanies or follows famine. The influence of want or famine is sufficiently potent to override the influence of season, with the result of a more than usual even mortality from cholera during the whole year.

It has been remarked that during times of famine insects of various kinds are more than ordinarily abundant. This is particularly the case with mosquitoes if there is no water-famine, and with flies bred in the superabundant human and animal offal. Hence numerous wounds and sores, which commencing from mosquito bites degenerate in the debilitated system into foul ulcers, to which often the scorbutic taint adds virulence. Similarly, there are more numerous cases of *peenash*, or "worms in the nose", during a famine; *first* from the superabundance of flies, and *secondly* from the debilitated condition of the people. *Peenash* is in reality maggots in the nasal passages. The disease is caused by a fly entering the passage and depositing larvæ, which eventually become maggots. Anyone may daily notice flies clustering about the eyes and nose of natives, who take little trouble to rid themselves of the nuisance. During sleep, or weakness from disease or want, flies are more likely to enter the nostrils. Considerable difference of opinion has been expressed as to the variety of the fly causing this malady. But the fact is that several varieties of flies may cause it, for the maggots are of different description. The advanced stage of this malady is one of the most loathsome coming under observation. There is constant discharge of offensive matter, and occasional epistaxis, while the bridge of the nose gradually becomes depressed, then ulcerated, disclosing the interior of the nasal cavity filled with a seething mass of maggots.

Other diseases becoming more prevalent during or after famines are small-pox (although this was not the case in Madras), itch, and other skin-diseases (especially "gunj" or scald-head), aphthæ, affections of the mouth, ophthalmia (especially ulcer of the cornea), and guinea-worm.

What I have said with reference to the maladies of the famine-stricken receives corroboration from Dr. Parke's observations on the condition of the men of Stanley's expedition after suffering from want of food. Parke writes: "Our men are getting extremely weak. In this debilitated condition, they suffer from ticks in the nose, and from gastro-intestinal catarrh; but by far the most formidable malady is the rapidly-spreading ulcers which form at the seat of the very slightest abrasion of the skin."

Sterility is a consequence of famine. It has been noticed that very few of the women in a famine-camp are pregnant; and it is some time after a famine that the normal birth-rate is reached.

I have a few observations to make with reference to suicide. A remarkable difference between English and Indian suicide is, that while in England the suicide death-rate among males is very nearly three times as high as among females, in the different Indian provinces the female suicide-rate much exceeds the male rate. Over three-fourths of the Indian female suicides drown themselves. An Indian female often commits suicide on very slight provocation. Indian females are emotional, prone to sudden bursts of passion, and often ill-used by the men. But it is a remarkable fact that suicide does not appear to be more common during seasons of want and famine than at other periods; yet the misery the famine-stricken endure is sufficient to justify suicide, if anything is sufficient to justify it. It may be that the reason why suicide is not more common during famine-seasons is, that there being little or no water, the favourite method of at least female suicide is not always available; for "the sight of means to do ill deeds makes ill deeds done".

Many Indian officers have had great experience in the management of famines, which is no easy matter, even when the resources of a Government are available. It is recently reported from Russia that the local administrations are quite unable to cope with the situation. At first sight, it does not appear difficult to assemble the people in camps, and put them to some famine-work; but the management of a famine-camp is as onerous as the conduct of an army. The people must be sheltered in some way. Then there is the important work of the sanitation of the camp, the people probably having no care for, or knowledge of, such matter. Water must also be provided. Then there is the police of the camp; robbery, peculation, and malingering have to be guarded against. A hospital must be provided, and medical treatment of the sick instituted. Then there is provision for the aged, infirm, and sick, unable to leave the villages. The idea which prevailed, and probably now prevails, amongst some civil officials and the public generally—that if a person can take food he does not die from starvation—has also to be combated. Surgeon-General Cornish, C.I.E., Sanitary Commissioner for Madras during the famine there, well demonstrated this necessity. He wrote to Government: "I have often

been assured that such and such a person did not die of starvation, because he was able to swallow food; but every physiologist knows that the human body may be starved of every essential necessary to its vitality, in spite of the ability to convey food into the stomach . . . . . Medical science recognises two distinct forms of starvation: one, in which food and drink is absolutely withheld; the other, in which daily food is insufficient in quantity or quality for the nutrition of tissue, and for the maintenance of the temperature of the body. But a great many officials and others still use the term starvation, in its restricted meaning, as applicable to what I designate the acute variety of starvation."

The moral of this is, that the recognition of famine ought not to be delayed too long. Governments are naturally desirous there should be no famine, for it involves great outlay. Hence the recognition of famine has been too long delayed in various countries. If I recollect rightly, although the register of births and deaths is very imperfect in India, still this register enabled Cornish to warn the Government long before the famine was officially recognised.

Another fallacy which has prevailed in some quarters is, that the amount of food which will sustain a man when at rest will sustain him when at work. When starving masses of human beings have to be kept alive, all questions should sink into insignificance in comparison with the vital one of how the largest number can be prevented dying at the least expense. And this question turns essentially on the suitability of the diet available.

In the *Report on the Progress of Hygiene A. M. D.*, 1876-77, it is stated that the Famine Commissioner sent by the Government of India to Madras "assumed that it was possible to feed the people on a smaller amount than was given in Bengal, and accordingly only one pound of grain was given, and this principally rice, instead of 1½ lb. with one anna for the purchase of condiments. This amount was hardly sufficient to maintain life in a state of rest, but was utterly inadequate for even a moderate amount of labour. The result was, that task-work being imposed, people perished by thousands." The question was most ably and vigorously discussed by Surgeon-General Cornish, whose views, fortunately, ultimately prevailed.

The minimum of grain allowance for a man in health and at work is not less than 24 oz.; while from 44 to 48 oz. is very commonly consumed by the natives of India, as any

one may readily imagine who has seen some natives' abdomens after their dinner.

In dealing with the famine-stricken several kinds of diet are necessary: viz., for labouring people; for non-labouring; for weakly; for sick; for children. Brigade-Surgeon Lyon, in his *Indian Medical Jurisprudence*, says that an adult requires *per diem*, if at rest, 25 grains, or, if at work, from 30 to 50 grains (according to the amount of work done) of nutritive carbon per pound of body weight. With this nutritive nitrogen must be supplied in amount equal to one-fifteenth to one-twentieth of the weight of the carbon. The food must contain from one to three ounces of fat. The food must contain a certain amount of salts, *e.g.*, phosphates, from ¼ to ½ ounce of common salt must also be supplied. Also from three to six pints of water. But different articles of food vary in composition, and in India many do not eat meat. It therefore follows that no diet-scale can be generally applicable. Diet-scales should be constructed with reference to the kind of food procurable, and with reference to the people for whom the diet is intended.

All the grain-foods in India have been analysed—wheat, bajree, raji, jowaree or cholum, bajree or cumbi, rice, gram, maize, etc.—so that there should be no difficulty in constructing the necessary diet-scale, although this cannot be done by anybody. And here I mention another fallacy which has prevailed, viz., that, provided the person got a certain amount of grain, it did not much matter what the grain was; yet, irrespective of the varying nutrient quality of grains, a sudden change is detrimental. Feeding people on bajree who have been accustomed to eat rice, or the reverse, is very likely to induce bowel complaint. Every Anglo-Indian knows that if he suddenly changes his horses' grain, gives *mout*, for instance, instead of *gram*, the animal will probably get "gripes".

Three systems of famine-work have been practised in India, viz., task-work, piece-work, and a third system, in which the wage being fixed, the work exacted is to vary with the supposed capacity of the individual. The objection to task-work is that on a minimum wage men of large frame and physique are not able to perform as much work as those of smaller frame. Piece-work involves too much pressure on the reduced. The third system is the best, provided there is an officer present capable of carrying it out, while at the same time preventing malingering.

The Government of Madras ordered that those unable to do 50 per cent. of work were to be kept in camps for special

light work, but if they were able to work and declined doing so, they were to be expelled. Now of course there are malingerers in all famine-camps, but if what I have said with regard to famine apathy is recollected, it will be seen that special skill is required to distinguish those who can and those who cannot work, and that no hard and fast rule is applicable.

The system of giving famine-works to contractors is to be deprecated. Contractors have their money to make, and must obtain good work from labourers. Public works officials are quite ready to demand the fullest amount of labour. This has been recognised in Indian famines, for under professional supervision the amount of food or pay supplied has usually been larger than when the supervision was civil or non-professional agency. In some parts of India the plan of giving the people food has been adopted on relief-works. In other parts the people were paid daily a sum considered sufficient to enable them to procure the necessary nourishment, which was supplied by grain-dealers and others brought to the camp for the purpose. There are objections to either plan. If money is plentiful prices will rise. Neither will the people always spend their money in the most desirable manner. In Russia it is complained that they purchase *Vodka* instead of food. Also grain-dealers are apt to adulterate the grain, as reported now from Russia. On the other hand, if the authorities purchase large quantities of grain, trade is disorganised, and, moreover, the authorities cannot purchase so cheaply nor transport so economically as the grain-dealers. The best plan is for the authorities to hold a certain quantity of grain, and if prices rise above what may be considered the legitimate market rate, the authorities should then commence giving grain. The grain-dealers, knowing this, will, in their own interests, take good care that the rise shall not be exorbitant. Local circumstances should, however, in a great measure guide action. And usually the Government of India, recognising this, have given discretion to the local authorities.

I here remark that in famines the fact has not been sufficiently recognised that growing children do not bear privation so well as adults, and require more nourishment weight for weight of body.

In most countries when famine occurs grain-riots take place. This has been recently the case in India. The people are prone to believe that the famine is due to the operations of the grain-dealers. Grain-dealers are not

always above making honest pennies from the misfortunes of their neighbours. It is a remarkable fact that, after the bunniahs' shops were looted at Cuddalpoore during the Madras famine, the price of grain fell considerably. There is another necessity and expenditure attending famine which has not been so generally recognised as the want of food. This is the want of clothing. Unable to procure fresh clothing, the people, especially the women, soon have scarcely a rag to cover their nakedness. Both decency and health demand that clothing should be supplied to the poor famine-stricken people.

I will now make a few remarks regarding the conduct of famine in Russia. It is reported from Russia that the local administrations are quite unable to cope with the situation, reckless abuses in the purchase and distribution of grain are heard of on all sides, and with so little knowledge is the relief distributed, or so badly is it administered, that the comparatively well-to-do are receiving unneeded help, while the really poverty-stricken peasants are dying of hunger. When the Czar gave instructions for the distribution of relief to the famine-stricken districts the officials were unable to carry out the order because the Government granaries had been robbed. In Russia the humblest official asserts his right to a bribe with as uncompromising a bearing as the chief authority. The authorities, both imperial and local, are utterly lacking in the knowledge of how to distribute relief so that it actually reaches the people for whom it is intended, and thousands of poods of grain are daily finding their way into the hands of what the *Novoe Vremya* calls the blood-sucking middlemen, and the bowelless *Koolak*, or village usurer. The only gleam of satisfaction emanates from Southern Russia, where the Red Cross Society began house-to-house visiting, relief in kind being distributed daily. Now I do not mean to say that mistakes have not been made in India, but I do mean to say that mismanagement in India has never assumed the proportions reported from Russia. The Government of India declared that no person should, if it could possibly be prevented, be allowed to die from starvation. And the Government officials, being English gentlemen, supplemented by Indian gentlemen, have nobly endeavoured to carry out the orders of Government. Doubtless numbers in India have died from starvation. But when famine is recognised as existing, relief is at once rendered available to all. From what I have said it will be abundantly evident that famine cannot be satisfactorily

dealt with unless sanitary medicine be brought into requisition to the fullest extent. There has been a tendency to ignore this, but it is nevertheless a great fact.

I have now a few words to add on the prevention of famine. If the Indian Government, or any other Government of a country liable to drought, seek to prevent famine, it must apply its energies principally to increase in the food-supply, and to extension of the means of distribution. Increase of food-supply is to be compassed by extended and better cultivation. Much has been done in this respect in India by encouragement given at fairs, agricultural shows, etc., by prizes, etc. Another means by which the food-supply will be increased is by the forest conservancy of recent years; for there are many parts of India where disforestment has resulted either in less rain, or in the rain flowing away, and not sinking into the ground. Then the endeavours made by the Government of Lord Mayo to further utilise fish as food should be persevered in, notwithstanding Mr. Hutchinson's idea that eating fish causes leprosy. But it is by irrigation that the food-supply can be most increased. At present, there are nearly 13,000,000 acres of land irrigated from canals in India, the works estimated to have cost 32½ millions of rupees. This, however, is not sufficient. It is not everywhere that there are rivers from which irrigation-canals can be extended. In such localities more wells are required, and in hilly districts more storage- and irrigation-tanks. But greater care should be taken that in irrigating the country it may also be drained, and that there is not over-irrigation and profuse waste of water. Drainage and irrigation are not incompatible. If they are not hand in hand, the country becomes waterlogged and unhealthy.

For the purpose of the distribution of grain, more roads and more railways are required. In the whole of Hindustan in 1890 there were only 16,277 miles of rail open, and 2,272 sanctioned. Then narrow-gauge rails were laid down where they ought to have been broad—the Rajpootana State Railway, for instance; and this notwithstanding the strenuous opposition of the local authorities at the time. In most countries, when famine occurs, there is plenty of food somewhere; but, for want of means of communication, it cannot be moved to the people.

Next, I have a word or two about rain-making. I have read several accounts of the production of rain by explosion of gunpowder, dynamite, etc. For example, I read a week or two ago, the following annotation: "Successful

Rain-Making.—The New York *Commercial Advertiser* made a successful attempt to bring rain to the Croton Aqueduct, near New York. A balloon was sent up into the clouds containing 200 lbs. of dynamite, which exploded with tremendous force. The explosion was immediately followed by a heavy downpour of rain.—*Dalziel.*" And in America a rain-making company has been formed. Now I believe it is quite possible to produce rain if there are clouds. Clouds are masses of minute vesicles. The liquefaction of vapours is their passage from the aeriform to the liquid state. Liquefaction may be due to three causes—cooling, chemical affinity, compression. When an explosion occurs, compression results; minute vesicles of moisture coalesce and become larger drops, and then they fall. If this be a correct explanation, some benefit may result from rain-making; but it would never suffice for the cultivator's purposes. Moreover, in some parts of tropical countries, when the rain fails, there are no clouds, the sky remaining, as I have seen it, all through what should be the rainy season, as if made of brass. In such times there would not be sufficient moisture in the air to be affected by explosions.

I previously observed that in a country subject to drought the Government should apply itself to extension of irrigation, etc. Fortunately, this country is not subject to such droughts as entail famine. But this country does not at any time support its population from its own agricultural resources. If war broke out, the coasts would be blockaded, and there is little doubt that the food-supply would be so diminished as to cause great scarcity, if not actual famine. The safety of this country from the squeeze of starvation during war depends entirely on naval supremacy, and this supremacy can only be obtained by making adequate provision during time of peace.

WHAT IS EFFICIENT VACCINATION?

By Brigade-Surgeon ROBERT PRINGLE, M.D.Edin., late Sanitary Department H.M.'s Bengal Army.

(*Read: January 20th*, 1892.)

Of all the known systems for the prevention, as opposed to the treatment, of disease, no single one has conferred greater benefits on humanity than the system known as vaccination, which may be described as the prophylaxis of small-pox; and thus no more important question could be asked than the one I have selected for the title of this paper, and I trust I may succeed in throwing some light on this subject, from a purely practical point of view, as opposed to conclusions mainly based on statistical data. For this purpose the correspondence lately carried on in the pages of *The British Medical Journal* has furnished me with ample matter for this communication, as regards what is considered in this country as "Efficient Vaccination". In the entire correspondence, statistics as regards numbers, sizes, and character of the vaccine cicatrices seem to be the bases of all conclusions, and not the results, as seen in immunity from small-pox, not with reference to individuals, but communities, like that of Sheffield, where a distinct system of vaccination has been practised for years, and its character guaranteed by large annual rewards for excellence. Take, for instance, the closing letter of this correspondence, viz., that signed "Herbert Goude", without date, which appeared in the issue of *The British Medical Journal* for Nov. 21st, 1891, where we read: "Finally, I would contend that the theory, that the protection from death from small-pox is afforded in an equal degree from vaccination in one place, as in four places, cannot be deemed to be worthy of consideration, until far more definite and complete evidence is put forward in its support, than has yet been elicited"; and it is in the hope of supplying this "far more definite and complete evidence", which Mr. Goude calls for, that I would place the following facts on record in the *Transactions* of the Epidemiological Society of London, on this very important subject.

If we are to accept Mr. Goude's theory, based, I presume,

on Mr. Marson's statistics; and his own experience in the Small-pox and Vaccination Hospital, Highgate Hill, no allowance whatever is made for the great variety witnessed in the severity of the various eruptive fevers, when there is reason to suspect that the infecting media have been similar, which variety is generally accounted for by stating that not only does the susceptibility to infection differ in different individuals, but the type of the disease resulting from this infection differs also in an equal degree.

Let me illustrate this in the case of inoculation for small-pox, as met with in the Himalayas, where it has been practised from time immemorial, as may be judged from the answer I received when I asked how long this practice had been known to them. The hereditary priestly inoculator pointed to his plough, and said, "As long as we have used that." In this instance, in 1865, I carefully examined the work of an inoculator, who was inoculating in one village, while I was vaccinating in the next, with the view of testing the merits of the two systems during a severe epidemic of small-pox. For six weeks the village in which the inoculator was working was kept in the strictest quarantine, while the villagers in the one in which I was vaccinating were allowed the freest intercourse with the villages around. When the quarantine was removed, I went to the village, and saw all the survivors, between sixty and seventy, of this inoculation, but found that ten per cent. had died, while some were very seriously ill, and, if they recovered, would be permanently disabled; but what interested me most was the fact that in no two cases apparently was there any similarity in the severity or mildness of the attack, and yet they were all inoculated *at the same time* with variola virus from *the one case*, in the form of powdered small-pox crusts, mixed with flour and "goor", dark treacle. The application of this compound was similar in every case, except that in the males it was at the base of the right thumb, while in the females at that of the left. The cicatrix, owing to the manner in which the virus was applied (viz., over a surface about the size of a sixpence, which had been almost denuded of skin, by repeated scratches with a bundle of five or three needles, tied together), in not a single case presented a trace of anything characteristic of a variolous cicatrix, and what was to be seen resembled more the characterless eroded surface, witnessed where variola had been severely confluent. This, no doubt, was due to the virus being fastened on with cotton-wool, until often excessive suppuration had taken place.

Now, in my opinion, the results seen in this village clearly proved that the resulting variolous eruption, and thus, according to the number-of-mark theory, the amount of imparted protection, has nothing to do with the number of places of insertion (as all were precisely similar, viz., one place of insertion), but with the susceptibility of each individual case, as regards the extent of the resulting eruption, therefore one, not even externally characteristic mark, affords as much protection as ten, or more; for surely no one who knows anything of the subject will compare the force of the contagion to be resisted in India with what is met with in this land of isolation, and small-pox hospitals, and all this without any thought of reinoculation. After a further study of the practice of inoculation, as seen under other inoculators during the years following 1865, I found that the inoculator under notice was not only an unskilful, but an unscientific operator, and as all the knowledge they acquire is hereditary, and limited to that of their father, the great variety of results in the practice of different inoculators is thus fully explained; but as I shall have to allude to that hereafter, I shall leave the details till then.

Here, perhaps, I may lay down what, in my opinion, constitutes "Efficient Vaccination". Efficient vaccination is a system of vaccination which requires neither revaccination, nor isolation of any kind, to secure an immunity from an attack of small-pox, similar to that experienced by a previous attack of the disease, whether naturally or artifically produced.

To illustrate, and thus to prove this statement, I shall allude to my own vaccine practice in India, in the Bengal Presidency, from Juggernauth on the Bay of Bengal, to Gungootree in the Himalayas, and shall begin with the experience of my own case. Soon after my arrival in India in 1854, seeing the prevalence of small-pox in Orissa, I looked for my own vaccine-mark, and could find none, though I was, I believe, vaccinated as a child, and I know I was vaccinated when eight years old, yet not the faintest traces of either of these operations were visible in 1854, so I vaccinated myself in one place on my right arm, with Dr. Graham Weir's vaccine-needle, which I then introduced into India, but found it had been used in the Himalayas from time immemorial. This cicatrix is now as clear and characteristic as it was thirty-seven years ago, and has protected me during small-pox epidemics, such as those who have only seen this disease in Europe can have no idea

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of. Surely on one would term this operation revaccination, without the faintest trace of any primary operation. Further, according to the number-of-mark theory, I have only one mark, but then, in that one mark there are clear evidences of nearly ten vesicles—a fact, by the way, strangely ignored when marks are alluded to.

Before proceeding further I had better take up the subject of revaccination, as, in my opinion, any system of vaccination which requires revaccination is perfectly useless for India, putting out of the question, for the sake of argument, the unphysiological aspect of the question, as why should the analogue of variola, viz., vaccinia, be the one exanthemata, the protective power of the primary attack of which should wear out after a few years, until by seven years it is so totally lost as to require artificial repetition.

In 1864, when I inaugurated this system of vaccination in my circle in the North-West Provinces, among a population of ten millions crowded together to the extent of 500 to the square mile, with a birth-rate of 60 in 1,000, the vaccine village returns, with reference to the revaccination column, had a foot-note, stating that the object of this repetition of the operation was to ascertain if the protective power of the first operation were still present. I at once saw, from questions put to me by the natives, that the presence of this foot-note would only tend to shake the faith of the natives in the prophylactic, and I accordingly suggested to the then head of the Vaccine Department in the North-West Provinces, the late Surgeon-Major F. Pearson, that the foot-note should be expunged from the returns, at least from those in my circle; for whatever the other Superintendents of Vaccination might do, I would never revaccinate, when I felt that the birth-rate was so partially even touched by primary vaccination. Though the revaccination column remained in the returns, the foot-note was expunged, and my circle alone presented the appearance of this column blank for the twenty years it was under my superintendence! It was hardly to be expected that this action on my part could prevent me from being a marked man in the Vaccine Department, and not of the North-West Provinces only, but well had it been for Dr. Pearson's work, and that of some of the other Superintendents of Vaccination, had they decided to have nothing to do with this revaccination, for it opened the door to an organised system of false returns, extending over years, till they were exposed by me in 1879-80, when the Government of the North-West Provinces in 1883-84 placed the following on

record, among other severe comments, regarding those of the second circle, that "it was then impossible to say to what extent the population of the circle (about 17,000,000) had been protected against small-pox", because, "in the Kumaon division also a practice of returning secondary vaccination as primary was discovered." In short, to such an extent had these false returns attained, that the Government of India, in a public document in 1884, stigmatised this vaccination as a "wholesale fabrication of returns", and, for aught I know to the contrary, unless some very radical change has come over the whole system since I left it in 1884, if this revaccination is still persisted in, things may be no better now.

During the practice of the revaccination to which I am alluding, the following vaccine phenomena were witnessed! When the rate of success was very high in the revaccination column, the remark was made that the primary operation could not have been good; and when, to meet this charge, a change of front was made, and the rate of success fell, then it was said, with the success so low, what need of revaccination? All this may seem incredible, but anything is possible in India, and, indeed, so utterly were the vaccine returns of the second and third circles vitiated by this revaccination, that I consider them useless as regards their exhibiting either any benefit from vaccination at all, or furnishing any reliable data whatever. For twenty years I pursued my own plan, and retired without the faintest recognition of my long service in the Sanitary Department; after my retirement, however, I enjoyed to the full the happiness of knowing that my system was completely successful, and my reward, *compulsorily obtained*, though late in arriving, was all I could have wished, and which came about as follows.

During the fearful epidemic of small-pox in 1884, which carried off, notwithstanding the vast numbers which died in the previous year, upwards of 200,000 in a population of 44,000,000, the immunity from the scourge of some of the districts in my circle (the first) was such that "the Army Sanitary Commission, in noticing the remarkable immunity enjoyed by the Meerut district, suggested that the cause might be specially investigated". This was done, with the following results: "In a letter which has been separately communicated to the Government of India, you have supplied the report called for, which corroborates the views already expressed by this Government, that the chief cause of the immunity is the degree of success attained by the Vaccination Department."

The Vaccination Department, forsooth—for it must be remembered that this special investigation was conducted, and the report written, after my retirement by a Department entirely opposed to my system of vaccination, and smarting not only under the reproof of its work having been stigmatised as a system of "wholesale fabrication of returns", but of my having been the means of exposing it. I had never revaccinated a single native during the twenty years from 1864, and had forbidden my subordinates to do so, and it is with evidence such as this that I can lay down, with I trust pardonable pertinacity—for I have suffered not a little for maintaining my views on this point: indeed, I threatened to resign the Department if obliged to revaccinate—that revaccination is *not* necessary. During the small-pox epidemics of 1883 and 1884 the force of the contagion must have been such as to most severely test the protective power of a *single vaccine operation on each arm*, and having stood that successfully, I feel I am justified in stating that revaccination is not necessary.

What therefore was the secret of this "Efficient Vaccination"? Like producing like, and nothing else, as met with in arm-to-arm vaccination. The very fact of the eruption producing its exact like *ad infinitum* is the best, as it is the only true test of complete (as far as the term is possible) protection. No spurious vaccination can produce true vaccination, and if any doubt exists, the first operation with the product clearly proves this. The practised eye tells at once the character of the vaccine lymph in use, not by the appearances seen on the seventh complete day, as by that time injury, dirt, etc., might render the eruption such that its true character might be completely changed, but by the appearance on the fourth or fifth day. Given a true period of incubation, and a true papular stage of the eruption, the test of the character of the eruption lies not in the vesicle of the seventh day—for that, as said before, is often injured—but in the early stages. As regards the necessity of revaccination, it may be interesting here to point out that true vaccination, as known to the Hindoos hundreds of years ago, was an operation that required no repetition; and to show that even the great Jenner was forestalled, as was also M. Pasteur, in India, I may quote here from an old Sanscrit document:

"The small-pox produced by the fluid from the udder of the cow will be of the same gentle nature as the original disease. . . . . The pock when perfect should be of a good red colour; filled with a clear liquid, and surrounded by a

circle of red; there will then be no fear of the small-pox as long as life endures."

The allusion to specially cultivated variola virus, according to M. Pasteur, is found in the following extract: "A certain quantity of cotton to be wetted with the matter of a favourable small-pox", and applied to an abraded surface on the arm. By "favourable small-pox" is meant a mild case, and undoubtedly variolous lymph thus selected produces, as I shall point out after, in the vast majority of cases variola *sine eruptione*, except at the place of insertion, just as in vaccinia.

To return to the tests for "Efficient Vaccination". Now let us suppose my vaccine-work, which stood such a test as that in 1884, had to be examined according to the rules of the Vaccine Department in Britain, not only would it have been considered most unsatisfactory, but probably called useless, and these vaccinators would have gone unrewarded; and yet, who that knows anything of vaccination would compare such work with that witnessed at Sheffield, for instance, and for which over £1,000 was given in rewards in eleven years? And what is the reason of this?—a very simple one, viz., that, owing to flies, dirt, and injury, very few of the vesicles, even excepting those from which the lymph was taken for carrying on the work, were allowed to heal up well, and thus the characteristics of a true vaccine cicatrix were lost, as in inoculation; but this has nothing to do with the protection afforded by the primary attack of the exanthemata, of which it was the evidence; that was seen and proved in the period of incubation and papulation; the rest follows as a sequence, and cannot possibly impart any protection. What therefore produced the unsatisfactory results of the vaccination at Sheffield —for if my line of argument has anything in it, the fault cannot possibly be due, as stated, to the neglect of revaccination? No, the fault lies in the amount of stored lymph employed, and chiefly no doubt the use of a semi-fluid subtance, or a pomade called calf-lymph, extensively made use of now. There is far too much irritation now, and too little vaccination; far too much appalling ignorance about the whole subject, and too much trusting to statistics; due, I believe, entirely to the complete absence of any means of studying the vaccinal eruption. Whoever heard of studying the eruption of any of the eruptive fevers by seeing the eruption only on the day it was complete? Why, let anyone see the vaccinal operation performed at some official stations; there is generally far too much blood drawn, and though the operation, owing to the small amount of true lymph

necessary for success, is remarkably successful, yet when thus performed it is wanting in skill, and undoubtedly frightens parents and children. I speak strongly on this point, because in inaugurating my system of vaccination in my circle, one of the objections to the operation was that it was a design of the Government to detect the black child with white blood that was to rule the world and turn the British out of India. My answer to this was, that any vaccinator who systematically drew blood would soon lose his appointment, and my vaccinators were all taught that, by tearing the skin by stretching it between the fingers, after slightly scratching it with the vaccine-needle on the arms of the children, they would obtain the best place for inserting the lymph, and would thus draw little or no blood.

All this may seem entering into trifling details, but it is attention to these trifles that leads to success, either in a voluntary system as mine was, or a compulsory one as it is here, by carrying the parents with us; and when it is thought what I was contending against—viz., a practice which was really a religious observance, as inoculation was considered, or a calm acceptance of the wrath of the offended goddess of small-pox, "Seetla" by name, to be appeased by gifts and ceremonies, but not by vaccination—the value of these details will be evident. Suffice it to say, my system, a purely voluntary one, with, at first, no aid and some opposition from the Civil Authorities, won its way by sheer force of conviction, and this even in the home of inoculation, Tirri Ghurwal in the Himalayas. The Maharajah of that country had his own son and heir vaccinated, and prohibited inoculation by infliction of heavy fines, and yet this was a system of inoculation so scientific that, as said before, in its practice M. Pasteur was anticipated by centuries, and the success of the system was simply perfect, indeed far superior, even with the slight risk of spreading the disease, to vaccination with revaccination. The system was as follows: By selecting the very mild cases for supplying the variolous lymph, the inoculator succeeded in imparting as perfect a protection from small-pox as it was possible, and this, with but rare exceptions, without any variolous eruption. This system I found among a certain class of hereditary priestly inoculators, men who had a very large and lucrative practice, and when they would enter the Vaccine Department, I gladly accepted them or their sons. This system required no reinoculation, and I repeat, the folly of attempting to supplant a scientific system of inoculation such as this

without repetition, by a system that was supposed to require renewal every seven years, or when small-pox was prevalent, was such that I would not be partaker in it.

The Royal Commission on Vaccination now sitting can never arrive at any true or satisfactory conclusions regarding the protective power of true vaccination, until they have seen what small-pox is unchecked by vaccination or inoculation, or what truly scientific vaccination is, and the test it has to stand. The greatest enemy to vaccination is its supposed simplicity, and next to this the introduction of calf-lymph, the production and collection of which exhibits, to my mind, a positive retrogression in scientific research appalling to contemplate. Look at the mode of extracting this lymph from an eruption very slightly vesicular, and then on the compound known as vaccine-pomade. *O tempora, O mores!* a return to the days of powdered small-pox crusts with treacle, and flour, or the still older ones of powdered crusts blown into the nostrils, in much the same way that those pests of civilisation, large small-pox hospitals, disseminate the virus now.

The only hope of attaining efficient vaccination in this country lies in a special department, where facts, not theories, are the bases of work, and where infinitely more care is devoted to studying the subject. To see, as I have seen, lymph boiled in closing tubes, and blow-flies resting on charged vaccine-points; to read of tubes of lymph sold at so much for a half-tube, and so much for a full tube—in short, to see and know all this is to feel confident that truly efficient vaccination on a large scale is almost impossible. I have carefully studied the whole question of calf-lymph, indeed practised it for a short time, and have come to the conclusion that, if the vaccinal eruption on the calf is to be treated like that on the child for the extraction of lymph, the calf-lymph practice must die a natural death; for all who have studied the subject know perfectly well, that the vesicular stage in the eruption on the calf and that on the child differ to such an extent, that, while pressure on the base of the eruption in the child's arm is *strictly forbidden*, unless a series of hideous clamps were applied on the poor calf's abdomen, lymph for only a *few cases* could be obtained. Surely the ominous silence of the German Vaccine Commission on this point should arouse suspicion. Again, what becomes of the lymph from the cases vaccinated direct from the calf with but little lymph in the inflammatory serum, yet this latter as fresh as it can be? Surely, if a promise has been

given not to withdraw any lymph, the crust, uninjured, might be obtained, and scientifically used, not as a pomade, but simply when deprived of all but the clear lymph in the centre, finely powdered with a little pure water, and applied, it will prove most successful; only do not let it be lost in these days of *pure vaccine-lymph scarcity*. I have stated before, in my definition of efficient vaccination, that, in addition to requiring no revaccination, it requires also no isolation to protect the vaccinated. This may seem a very startling statement, but it is one with which we are all quite familiar in India.

As I said before, the system of vaccination which I carried on was purely voluntary, and thus it was only those children whose parents brought them for vaccination who were operated on; but, of course, there were numbers in the village who were unvaccinated, and it was among them that small-pox was almost an annual visitor, and this was the contagion my vaccination had to meet annually, and met it successfully. Little wonder therefore that I am such an enthusiast in the matter of efficient vaccination. What would the Vaccine Department in this country say if, with nearly every full moon from April to October, under one of the large trees in one of our public parks, hundreds of women should assemble with their children, many of the latter covered with small-pox in every stage of the eruption, to propitiate the goddess of small-pox, and, mixing freely with them, numbers of vaccinated children—or suppose the children in the families attached to some of the large laundries near London were to be suffering from small-pox, and to be found playing among the clothes ready to be sent out—or, to come nearer home, suppose your servant, on hearing she is called by her mistress, suddenly puts downs her own child covered with small-pox, and takes up your own with as little concern as if there were no such thing as infection? Vaccination that will not successfully resist this test *without isolation* is useless for India, and I maintain the same for Britain. What is capable of attainment in India is surely so in Britain, the home of Jenner, but it will never be under the present system. When vaccination is laughed at by Leicester, and mourned over at Sheffield, when anyone can sell what he calls vaccine-lymph under conditions which makes it worse than harmless, and this in a country where butterine was by Act of Parliament called margarine, in case imperfect enunciation might lead to a confusion in the name, and a compound *mysteriously made* being sold

for the true and simple product of cow's milk. I repeat, when all this is in our midst, it is time some action was taken. When I commenced vaccination in 1864 in the North-West Provinces, and was working in the villages practising female infanticide, the little girls were freely brought for vaccination, as one of the reports which got abroad was that the object of the Government in giving this *vaccination free* was with the view of reducing the surplus population, and these villagers thought they might lay the deaths of their little girls at my door, as the saying is in India; but their surprise was great when these girls escaped the scourge of small-pox, and their boys died. This changed all, and before I left India I witnessed the following in many of these villages, viz., the girls left unvaccinated to die of small-pox, when no questions were asked by the police, and the boys protected from small-pox by vaccination! So palpable was this in some villages that I reported them specially to the Government.

Only once during my Indian service of thirty years was my vaccination called in question, as failing to impart the protection from small-pox which I claimed for it, and this was in the city of Meerut, the circumstances connected with which are so instructive that I allude to them in detail. When in camp near Agra I received a letter from the Magistrate of Meerut, reporting that the natives in the city were complaining that the vaccination they had accepted for years had failed that year to protect them from small-pox. I at once decided upon visiting Meerut, and there, sure enough, the failure was explained by the presence of an epidemic of varicella. It would have been of little use to explain to the natives, that the two diseases had nothing in common, and that vaccination afforded no protection from chicken-pox, which they erroneously called "little small-pox", unless I could satisfactorily illustrate this to them; and here, owing to the voluntary character of the scheme, some cases of small-pox were met with, and having collected the head-men, I showed them the difference by means of the following simple illustration. Placing a case of small-pox alongside one of chicken-pox, I pointed out that in the latter there were crops of the eruption, like the various kinds of grain they sow in one field, and which rise and ripen at different times; in the other it was like a field of wheat only, all the same size, and ripeness. This completely convinced them, and vaccination scored a victory out of what seemed a failure. I proved the correctness of my diagnosis in my own

family, for I brought the infection of varicella to my house, and the only child that escaped was one who had suffered from the disease in Scotland. During this epidemic an isolated hospital-tent attached to one of the large general hospitals was marked "Small-pox Hospital", and I can certify that there was not one case of small-pox in it, a fact clearly proved afterwards, all being cases of varicella!

In conclusion, it is too clear, from the line that the evidence is taking before the Royal Commission on Vaccination, that the days of compulsory vaccination are numbered, and with such divisions in the camp of vaccination as the late correspondence on "Efficient Vaccination" has elicited, and the melancholy absence of practical as opposed to theoretical conclusions, which the arguments show, little wonder the opponents of vaccination step in, and their leader, Mr. Tebb, in a letter dated November 26, 1891, states: "One mark is now described *as imperfect vaccination,* and the imperfectly vaccinated die at the rate of 26 per cent., and so on. Yet it was upon one mark that the original successes were claimed to be won, and it is upon one mark that all the early proofs depend. However, be that as it may, the people having been so fearfully wronged by the outrageous Acts, are determined not to wait for the Report of the Royal Commission, but to elect representatives for the next Parliament who will promise to redress their grievances, and purge the statute-book of this most oppressive and mischievous legislation."

Thus I end as I began, with the *number of marks* as a test of protection, and efficient vaccination, and the consequences of this unscientific line of argument, viz., efforts to repeal the compulsory clauses in the Vaccination Acts, than which, with the faith of the nation considerably shaken in Jenner's great discovery, by the conspicuous failure at Sheffield, etc., nothing more calamitous could happen to this country; for with small-pox once epidemic, with our dense population and our miserable arrangements for vaccine lymph, we should be called upon to meet a scourge for which we should be totally unprepared, as the worst way of meeting a small-pox epidemic is a system of large small-pox hospitals. Let the money spent on these buildings be expended on a special department, and let all conspicuous failures in vaccination, such as that lately seen at Sheffield, be inquired into by special independent officers, and not by those who are practically reviewing a system, the rewards for the creditable carrying

out of which may in a great measure have depended on their own previous reports. When an accident happens in a railway system, the cause is searched out, not by the civil engineers of the line, but by Royal Engineers under the Government, specially trained for this important duty. So let it be in vaccination: let the public demand an independent inquiry into Government failures, as the Government do into Railway Company failures; then, but not till then, will facts triumph over figures.

IS INFLUENZA IN MAN AND IN ANIMALS ÆTIOLOGICALLY DISTINCT?

By RICHARD SISLEY, M.D., M.R.C.P.

(*Read: February 17th*, 1892.)

ALTHOUGH a comparative study of the diseases which affect animals and man has attracted the interest and the attention of the more thoughtful portion of mankind for centuries, the subject has received less notice than its importance merits. Medical studies are still carried on in our hospitals and in our universities with little regard to the question, and at the present moment no systematic instruction is given on it in any school in this town. The Apostles of Drugs still flourish, and the attempt to cure a disease which is best left to run its course is still more popular than a study of the natural history of the disease. The Epidemiological Society possesses the great advantage of bringing together, to meet on common ground, not only those who take an interest in epidemics amongst men, but those also whose chief care it is to guard against, and study the history of epizoötics in animals. From such a representative body I hope therefore that some new evidence may be forthcoming on the question, "Is influenza ætiologically distinct in man and in animals?" and I hope that future investigations may clear up some obscure points.

The question is beset with difficulties which cannot be passed over in silence. It cannot at present be confidently affirmed that we are fully acquainted with the ætiology of human influenza. It is generally, if not universally, admitted that the most deadly epidemics have had a foreign origin. Those of the years 1510, 1557, 1732, 1743, 1782, 1803, and 1837, all, to use the words of Dr. Peacock,* "extended their range from Asia through Russia and Turkey to the countries of western Europe". But in Europe sporadic and epidemic outbreaks occur of a disease which careful observers believe to be influenza.

* *On the Influenza, or Epidemic Catarrhal Fever of* 1847-8. London, 1848, p. 3.

Dr. Fiessinger* says that in Oyonnax—a little town of 4,000 inhabitants, situated at the foot of the Jura Mountains —influenza frequently occurs in bad seasons. In 1887 a number of children were affected. In January and February 1888 there was an epidemic.

In 1890 the Minister of Public Instruction of France appointed a commission to investigate influenza in Russia, and the result was the production of a valuable report written in the charming style possessed alone by French medical writers. In his report M. Teissier† clearly shows, as a result of inquiry on the spot, and by reference to official documents, that influenza is believed to be endemic in Russia.

Sir Peter Eade,‡ in 1878, described an epidemic outbreak in Norfolk, analogous to, if not identical with, influenza. In answer to a letter, in which I asked Sir Peter Eade what opinion he had formed as to this outbreak, I received from him the following reply, which I am sure will be of interest to the Society: "As to what you ask me about the epidemic of 1878, as observed in this district, it lacked much of the head and back pains, and showed rather more catarrh than our recent outbreak; but in all other respects it was similar. I formed the opinion that it was *modified* influenza." That catarrh is often called influenza appears certain; but the histories of the epidemics spoken of by Teissier, by Fiessinger, and by Sir Peter Eade, leave no doubt in the mind that these outbreaks are by no means to be explained by so simple an hypothesis. To what then were these outbreaks due? What is their ætiology?

This initial obscurity in the ætiology of influenza in man has an analogue in the ætiology of equine influence. Some veterinary surgeons, I am told, apply the term influenza to a catarrhal disease, which by others is called "London fever". In France MM. Megnin and Veillon§ came to the conclusion, after careful consideration, that until French veterinarians expressed more clearly what they meant by equine influenza, it was useless to draw any deductions from observations on the disease.

In England, however, there is fortunately no such hopeless ambiguity about the writings of our best veterinary

* *La Grippe infectieuse à Oyonnax (Ain), Novembre* 1888 *à Avril* 1889. Paris, 1889.

† *L'Influenza de* 1889-1890 *en Russe.* Paris, 1891.

‡ *Lancet*, March 10th, 1878. "Medical Notes and Essays; Fasciculus ii: Influenza." Jarrold: London and Norwich, 1891.

§ *Comptes Rendus hebdomadaires des Séances de la Société de Biologie, q. s.*, tom. ii, p. 180.

authorities, who have indeed been greatly in advance of most recent medical writers on human influenza. As long ago as 1875 Professor Fleming* clearly and strongly expressed the opinion that equine influenza was contagious; whilst it may be remembered that as late as May last, doubt was thrown, even at a meeting of this Society, on the fact that influenza in man is spread by contagion.

It is obviously impossible in the time at my disposal to enter fully into any historic account of the epidemics of influenza which have affected man, and the epizoötics of influenza which have affected animals. Nor is it possible, on the other hand, to entirely ignore the teaching of history.

The quantity of literature on the subject is appalling; a large library would not contain it, nor could any man in the course of a long life hope to fully master it. Fortunately, several writers have made it possible to get a general idea of the history of influenza in man. Dr. Frank Clemow† has made a special study of the earlier epidemics; the later ones have been summarised by Dr. Copland,‡ Dr. Theophilus Thompson,§ and others; and more lately Dr. Charles Creighton‖ has added considerably to the sum of our knowledge of the disease as it affected this country.

An exhaustive account of epizoötics of influenza in animals is given in Professor Fleming's classical work, *Animal Plagues.*

From these sources tables have been drawn showing the chief years of epidemics and epizoötics of influenza. History, as recorded by the authorities quoted and summarised in the tables, shows:

1. In some years epidemics of influenza have been unaccompanied by epizoötics in animals.

2. In some years epizoötics in animals have been unaccompanied by epidemics in man.

3. In some years epidemics in man and epizoötics in animals have co-existed.

It might be argued from this that there is no connection between epidemics of influenza in man and epizoötics in animals, and that the occasional coincidence of them is a matter of chance. What direct evidence is there pointing

* *A Manual of Veterinary Sanitary Science and Police.* London, 1875.
† *Public Health*, vol. ii, p. 359.
‡ Copland's *History of Medicine*, vol. iv.
§ *Annals of Influenza.*
‖ *A History of Epidemics in Britain from* A.D. 664 *to the Extinction of the Plague.* Cambridge University Press, 1891.

to the spread of influenza from man to animals, and *vice versa?*

First, with regard to horses. There is absolute unanimity amongst veterinary surgeons that horses have influenza, and that the clinical varieties of the disease are similar to those which occur in man. But as to whether influenza can spread from horses to man or from man to horses there is no agreement. Dr. Theophilus Thompson's book supplies no instance. Professor Fleming, in his experience and his research, has come across no such instance. In a letter he kindly wrote to me in 1890, and which with his permission was published,* he says, "Neither is there any evidence that the disorder has ever been communicated from horses to mankind, or *vice versa*, notwithstanding the great opportunities for such transmission, if such could be effected, which are offered in large horse establishments and in cavalry regiments."

This question was discussed at a meeting of the Lincolnshire Veterinary Association in February 1890, and there was a general expression of opinion that influenza did not spread from horses to man, and only one member expressed a different view—namely, that, to quote Captain Russell's own words, "men may contract a similar complaint from the emanations or issues given off from the animal (horse)." Let us see what positive evidence has been offered in support of the theory that influenza does spread from horses to men. The most careful and complete piece of evidence I have yet found is that recorded by Mr. Caird of Lochgoilhead.† His account of the outbreak, in his own words, is as follows:

"On January 2, 1890, two horses, aged respectively five and six years, were seized with influenza. The symptoms were as follows: short cough, profuse nasal discharge, nauseous breath, marked stiffness of joints, disinclination for food or drink; no lung complications; duration of illness about three weeks. . . . . From January 9, 1890, to February 10 a dancing-class was taught in the hay-loft above the stables. This loft was low in the roof, with deficient ventilation, and the infected stables below were in a most insanitary condition. There was direct communication between the mangers out of which the infected horses fed and the hay-loft, through which a current of air passed into the loft and contaminated the atmosphere.

* *Epidemic Influenza: Notes on its Origin and Method of Spread.* Longmans, 1891.

† *Lancet*, 1891, vol. i, p. 741.

There were forty people at the school, mostly the children of shepherds, isolated from each other over an area of about four or five miles from the hay-loft where this class was held. There were only about a dozen families in the district, and owing to their pursuits in life and primitive habits, they have little communication with the outside world. There was no case of influenza in the district or anywhere near it, as far as I know, before February 1, 1890. On that date a person, who was in continual contact with the horses, was seized with the disease. On February 2 his son and a neighbouring lad were attacked with influenza complicated with pleuro-pneumonia. . . . . All the forty pupils in the dancing-class suffered from well-marked influenza except seven. . . . . At Lochgoilhead, about nine miles from Carindow, there were five or six mild sporadic cases after the epidemic at Carindow, but there were no other cases, to my knowledge, within ten miles of Carindow." Mr. Caird's account of this outbreak is so circumstantial, and recorded with such care, that it cannot be ignored.

The Local Government Report* on the epidemic of 1889 and 1890 contains some interesting information on the same subject. On page 262 of the report a case is recorded in these words: "Mr. J. B. Gresswell of Louth has had a very large experience of horse-influenza. Since the spring of 1883, it had persisted in Lincolnshire. He has known of its special prevalence in Grimsby and district. In August 1889 Mr. Gresswell's four horses contracted the disease, he says from some infected horses in a stable, the ventilators of which opened into his yard. Shortly after his own horses were taken ill he himself was taken suddenly ill. His temperature was found to be 102°. He felt quite 'done up'. After a few days his acute symptoms subsided, and he resumed work, but had a relapse. This was followed for some months by great nervous depression, palpitation, and shortness of breath. His assistant, shortly after Mr. Gresswell's illness, was also attacked. He (Mr. Farr) describes his symptoms as at first consisting of headache, fever, great prostration, and a sense of having been beaten all over. This attack came on quite suddenly, and he remembers great aching in his limbs. He had several relapses, and suffered afterwards from shortness of breath and great palpitation. He did not finally get rid of his symptoms till he had a complete change of air and rest from work. . . . . Mr. Gresswell and his assistant are confident

* London: Eyre and Spottiswoode.

that their ailment was taken from the horses; many animals besides Mr. Gresswell's own suffered at the time; both the assistant and the principal were daily in contact with many infected animals. They were also greatly overworked."

These cases appear to be important. In August 1889, it will be remembered, there was, as far as is known, no prevalence of influenza amongst men. The report goes on thus: "Mr. Gresswell says he remembers three other instances where persons engaged in waiting on infected horses developed the disease."

But of these cases the writer of the report justly points out that one is open to objection, because at the time human influenza was prevalent in the district. Of the other two cases no particulars are given.

In the same report a case is given in which a doctor believes his horse received the infection of influenza from the groom. The case is as follows: "At Thirsk, Dr. R. Hartley informed me that both he and his groom had influenza (contracted from his patients) in December. Within a fortnight his best horse was unable to work and was pronounced to have 'influenza', while his other two horses were amiss and had bad coughs. Dr. Hartley is of opinion that the horses contracted their illness from the groom. He did not know of horse-influenza existing in the locality at the time."

The next question is, *Do dogs have influenza?* In his account of the epidemic of 1775, Dr. Fothergill wrote*: "During this time horses and dogs were much affected, those especially that were well kept. The horses had severe coughs, were hot, forebore eating, and were long in recovering. Not many of them died that I heard of; but several dogs."

During the same epidemic, Dr. William Cumming,† of Dorchester, wrote: "About the latter end of October, I remember to have heard that one gentleman had lost six dogs in the usual manner that these animals are seized—a giddiness of the head, an inability to eat, with a paralytic affection of the hinder extremities." And Dr. Thomas Glass, of Exeter, made a similar observation: "In the month of September", he says, "many horses and dogs were severely afflicted with colds and coughs." Dogs were also affected at Blandford, in Dorsetshire. Dr. R. Pulteney

* *Medical Observations and Inquiries by a Society of Physicians in London*, vol. vi, second edition, 1812, p. 346.

† *Op. cit.*, p. 359.

wrote: "I heard much of horses and dogs having been affected before we heard of it among the human race."*

Dr. Theophilus Thompson says that during the epidemic of 1803 the dogs of Dublin suffered from sore eyes; and during the same year Mr. Richard Dunning,† on the authority of Mr. Parsons, a farmer, near Modbury, says: "Dogs were generally affected with a disease, termed of them the Houst; which, he observed, seems to consist of a continual effort to vomit, and that froth and slime were thrown up by these efforts in considerable quantities; many of these animals were ill several weeks, and many died."

In 1889 some careful observations were made by MM. Megnin and Veillon,‡ at Chantilly and l'Aveyron, and these gentlemen came to the conclusion that the infectious malady from which the animals were suffering was analogous to, if not identical with, influenza.

More recently, Dr. Parsons and Dr. Bruce Low have collected evidence which is in favour of the theory that dogs suffer from influenza. These cases occurred in the Caistor Rural Sanitary District, Lincolnshire. The report contains the following passages: "I have obtained information from three of the principal farmers of this village regarding the prevalence of 'influenza' among cats and dogs."

"At the first farm, a valuable pet pug bitch was taken ill on December 2. Its symptoms were prostration, fever, ending in diarrhœa; it took no food for three days, and then quickly recovered. Its playfellow, and also a pet of the house, was a kitten. This animal was taken ill just before the pug (about December 1). It had similar symptoms to the pug, but had running from the eyes and nose; it recovered after a few days' illness."

In the second farmhouse, "at the beginning of December, a terrier dog who came into the house every day and played with the children, was taken ill, sick, feverish, hot nose, very weak and listless. It recovered after a few days' illness. This terrier was also accustomed to visit with his master almost daily the house mentioned, when the pug bitch was ill. At this date, the beginning of December, influenza was quite general among the people of the village."

At the third farmer's house, "early in December, a large retriever dog which ran about the yard and stables, was

* *Op. cit.*, p. 402.
† *Medical and Physical Journal*, vol. x, p. 137.
‡ *Bulletin de l'Académie de Médecine*, 3 s., tome xxiii, p. 115.

noticed to be ill; it had a hard, dry cough, 'like that of an old man,' its nose and eyes ran, it was feverish and looked very ill, it could hardly walk; its illness lasted only a week."

Do cats have influenza? On this question the evidence is perhaps more complete, and at least two recent cases are on record which are well worthy of consideration.

"At a meeting of l'Académie de Médecine (Paris), on January 28, 1889, M. Auguste Ollivier* related a case which seemed to him to prove that influenza could be communicated to a cat by matter expectorated by a human subject. A lady had a violent attack of influenza, which was accompanied by the expectoration of much viscid mucus. As she was extremely feeble, and took food with difficulty, her doctor advised her to suck pieces of meat. She extruded from her mouth several morsels which she had chewed, when a cat seized upon them and swallowed them. Three or four days later the animal died, having suffered from symptoms of influenza, namely, from cough, dyspnœa, and loss of flesh."†

The other case is given by Mr. Caird in the communication from which I have already quoted, and points to the horses as having been the source of infection. "The owner of the horses at Carindow", he says, "had three young cats which frequented a hay-loft over the stables where the horses were kept while suffering from influenza. These cats were often seen in the stables or about the stables where the horses fed. They were soon seized with sneezing, cough, discharge from eyes and nose, were disinclined to take solid or liquid nourishment, and suffered from severe purging. One died, the others recovered."

The Local Government Board Report, to which I have already more than once referred, contains evidence in favour of the hypothesis that cats have influenza. At the farm at which the pug bitch and the kitten had the symptoms previously referred to, "at the same time the groom's cat which lived in the stable was taken ill much in the same way, but recovered. Owing to the swarm of rats which have lately multiplied so greatly in the locality, the farmer kept five cats in the yard. These did not enter the house, but would of course be in communication with the house cats. Three of the yard cats died after a short illness, just before December 3, and the other two just after

* *Bulletin de l'Académie de Médecine*, 3 s., tome xiii, pp. 118-19.

† *Epidemic Influenza: Notes on its Origin and Method of Spread.* Longmans, 1891.

this date. The farmer's groom asserts that he can recollect the names of persons who among them in the village lost twenty-two cats about the end of November and the early part of December. He further states that in his opinion there were others of which he had not heard.* At a farm one mile from this village, three cats died in December after a short illness, and at another farmhouse, two miles off, two more died in a similar way, about the same time. All these people deny that poison could have killed the cats, since the farmers had forbidden the destruction of rats by poison, fearing that poisonous food might be devoured by their dogs, poultry, or other animals. Some also feared their children might touch the poison. In addition to the cats that died, it is said that, both in this village and those adjoining, many other cats were ill, but recovered."

At another farm, "during the last week in November, the farmer's two children, two servants, and a cat were all taken ill with influenza. About the 1st of December the groom's cat was also taken ill; it recovered, but the first cat died. Ten days later, another cat, which was brought from a distance to fill the place of the deceased cat, was taken ill and died on the fourth day of its illness. Its symptoms were, running from the eyes and nose, diarrhœa on the second day, fever and weakness. Latterly it could not walk."

Sir Peter Eade, in the instructive letter which I received from him to-day, kindly gives me the following interesting information:

"We know that (rightly or wrongly) influenza is said to attack horses. It may interest you to learn that a large pig-farmer told me the other day that he was sure many of his hogs had recently had the influenza. Those affected were seized with fever, panting, and distressing cough, and then in two to four days the illness appeared to pass off, and they were well, except for a little cough, which often remained a few days longer. One or two of them died."

Mention must be made of recent German researches on the bacteriology of influenza. Dr. R. Pfeiffer professes to have found the bacillus which causes influenza, and states that he has by inoculation given the disease to apes and to rabbits. These observations require confirmation; leaving

* The value of this observation is not apparent. Those who are unacquainted with Dr. Parsons's report will not act fairly if they form an opinion on it simply by reading quotations from that part which refers to influenza in animals.

them for the present out of consideration, it seems, then, that there is no doubt whatever that horses, dogs, and cats suffer from a disease similar to, if not identical with, human influenza.

With regard to the infection of human beings from diseased horses, the cases recorded by Mr. Caird and by Mr. Gresswell seem to me to point strongly to the probability that such infection may take place, and I am also inclined to attach importance to Dr. Hartley's case, in which he believes his horse was infected by his groom.

In the case of cats, M. Ollivier's and Mr. Caird's cases are also of great interest, and such evidence cannot, I think, fairly be ignored.

In conclusion, let me say, the evidence at our present disposal seems to me to point to the fact that although the spread of influenza to man from animals, and from animals to man, is certainly not the usual one, that there is sufficient evidence that both these ways of infection may occur to make the subject worthy of careful and thorough study.

BACTERIOLOGY OF VACCINE LYMPH.

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NUMEROUS have been the attempts, ever since bacteriology first took rank as an exact science, to solve the problem as to the nature of the active principle of vaccine lymph; and in consequence of this, and of the great difficulty of the subject, a mass of literature has gradually accumulated, sufficient to appal anyone newly entering upon the task.

The fact, however, that, in the absence of any sufficient knowledge on this point, the whole practice of vaccination as at present carried out is more or less empirical, may perhaps furnish excuse for still further attempts in this direction. I therefore venture to bring forward the results of an investigation of this subject, on which I have been engaged at intervals for the last eighteen months. And I am especially glad to have the opportunity of bringing forward here a paper on this subject, for the reason that the Epidemiological Society has always shown a deep interest in the subject of vaccination; such interest, indeed, dating from the foundation of the Society in 1850. In that year a committee was appointed, which carried out a most laborious and comprehensive investigation into the practice of vaccination in the United Kingdom and the British possessions; and the conclusions at which they arrived, of much value and importance, were embodied in Lord Lyttelton's Vaccination Act of 1853.

As a first step, it appeared desirable to obtain proof, if possible, of the particulate nature of the essential cause of vaccinia, since the theory has recently been advanced that this is to be looked for, not in the form of a living micro-organism, but in that of an enzyme capable of reproducing itself by means of its own action on the living tissues with which it comes in contact; the supporters of this view relying on the want of success that has hitherto been obtained in attempts at cultivation of the virus outside

the animal body in various nutrient media in justification of their contention; although, as far as I am aware, no trustworthy evidence has ever been brought forward by them in support of such a theory.

With the object, then, of putting this point to the test, I determined in the first place to repeat, under possibly more favourable conditions, experiments which had previously been carried out by Cohn, Chauveau, Burdon-Sanderson, and other observers; by whom demonstration had apparently been afforded that while vaccinia was incapable of being produced by the fluid portion of vaccine lymph from which all solid particles had been separated by deposit or filtration, on the other hand inoculation of such separated particles was capable of setting up vaccinia.

My own experiments were carried out in the following manner. With some difficulty about two cubic centimètres of calf-lymph were obtained, and intimately mixed with double that quantity of normal saline solution, or of a 50 per cent. solution of glycerine in distilled water. This dilution was carried out with the object of obtaining a more workable amount of fluid, experience derived from inoculation-experiments having shown that such admixture of dilute glycerine exerted no deleterious effect, but rather if possible the reverse, on the potency of the lymph to which it was added. An additional advantage was also found in the fact that the fluid obtained, in the latter case especially, did not tend to become of increased density from exposure to air during the process of filtration, as is liable to occur if pure vaccine-lymph is employed alone. Such diluted lymph was, for the purpose of these experiments, aspirated through a Kitasato's filter by means of an exhaust-pump actuated by water-pressure, the porcelain bougie resting in a small test-tube of but slightly larger diameter than its own, the test-tube itself being enclosed, for convenience of working, in a larger flask connected up with the pump. In this way extremely small quantities of fluid could be exposed to filtration-action, while loss of the filtrate was obviated by keeping it stored in the test-tube (previously sterilised) into which it dropped, and which was securely plugged and covered with an india-rubber cap as soon as sufficient had been obtained.

With the object of testing the potency or the reverse of the filtrate, a number of inoculations were carried out, both on human beings and on the lower animals, especially on calves, but in no single instance was any obvious result

obtained, no vesicle putting in an appearance at the seat of inoculation.

In this respect, then, my experiments confirmed those of previous observers; but although they might appear to prove beyond all doubt that the active principle of the lymph was indeed particulate, further consideration showed that this is not necessarily the case. For Sidney Martin has demonstrated, in connection with his work on the products resulting in nutrient media from the vital processes of the anthrax bacillus, that if any of the alkali-albumen in his artificial serum has failed to become converted into albumose, such alkali-albumen is separated off during the process of filtration through porcelain, to which the fluid is subjected by him with the object of removing the bacilli themselves. I have myself observed a somewhat similar fact. When searching for a good medium for certain organisms, it struck me that milk would fulfil the conditions I required, provided the fat-globules could in some way or other be separated from the other constituents without these latter becoming in any way altered. Thinking that possibly filtration through porcelain might have the desired effect, I put the matter to the test, with the result that I obtained a perfectly transparent fluid like water, but possessing a slightly yellowish tinge. On examining the filtrate chemically, however, I found that not only had the fat been separated, but the greater part of the casein also, while the fluid contained the normal proportion of serum-albumen, sugar, and salts. In this instance, also, a body somewhat allied to alkali-albumen, and in solution in the milk experimented upon, had been unable to find a passage through the pores of the filtering material; consequently, although the theory of a *contagium vivum* is all but universally accepted at the present day, it will be seen that from such experiments as I have described, which show that certain bodies of a proteid nature are incapable of passing through a porcelain filter, that such a method of treatment, when applied in the case of vaccine lymph, does not demonstrate with absolute certainty that, because the filtrate is incapable of inducing vaccinia when inoculated into the animal body; therefore, the essential principle of vaccine virus is particulate.

It may be objected that "deposit" experiments are not open to a similar fallacy; but it must, I think, be obvious that if the numerous bacteria present in vaccine lymph, many of which at any rate have in my belief no part in the specific action of the lymph, be separated in such

a manner, there is no reason why they should not carry down with them minute traces of some substance possibly of a proteid nature, which, as contended by Dr. Cory and others, may represent the essential principle of the virus. The outcome of my filtration-experiments goes, then, to show that, although the results are not incompatible with the generally-received opinion as to the particulate nature of the important constituent of vaccine lymph, the problem is perhaps not quite so easy of solution as might at first sight appear to be the case. And a similar remark will of course apply to experiments of a converse nature, in which removal and inoculation into the animal body of such constituents of the lymph as are retained by a porcelain filter has resulted in the production of normal vaccine-vesicles.

I next turned my attention to the identification of the various micro-organisms which can be grown as the result of inoculation of various nutritive media with vaccine lymph. And here I should remark that in all the experiments of this series calf-lymph alone was used, as it seemed probable that by this means one would obtain more constant results.

The number of experiments made by me for the purpose of cultivation of bacteria present in the lymph has been so great, that it would be wearisome to give any detailed account of them, and consequently I propose giving, on the present occasion, a *résumé* merely of the results that I have obtained.

In the first instance, both "streak" and "stab" inoculations were made with a trace of lymph on the surface, or into the substance, not only of nutrient gelatine, but also of agar-agar, and of glycerine-agar masses. In all of these, growth became apparent after the lapse of a few hours or days, according to the temperature at which the tubes were kept. The resulting growth was usually tinted of a more or less yellow or orange colour, particularly in "streak" cultivations, or at the upper part of those resulting from a "stab"; in other words, where there had been free access of air; and the colour was in every case much more marked in gelatine than in agar cultivations. The cultivation resulting from a "streak" inoculation was, as growth went on, seen to present distinctly raised or rounded edges, the whole mass being more or less glistening, and often not uniformly coloured. A "stab" cultivation, on the other hand, showed greater luxuriance of growth on the surface, where there was free access of air, than lower

down; but it usually occurred to a greater or less extent along the whole line of the needle-track, the colour of the growth, except at the free surface, being of a greyish-white, with occasionally the slightest trace of brown. In addition to the well-defined line, representing the path that had been taken by the inoculating-needle, numerous discrete points of growth were seen as minute dots extending beyond, but parallel to, the main line of development. Small portions of these growths, when transferred to cover-glasses and stained in various manners, showed masses of micrococci similar to, but of course in much greater numbers than, those which could in like manner be demonstrated in vaccine lymph itself.

As it was evident that more than one organism could usually be obtained by the inoculation of lymph into nutrient media, plate-cultivations were made with the object of separating out the different varieties present, both gelatine and glycerine-agar being used for this purpose. In this manner I succeeded in obtaining pure growths of various micro-organisms, of which those that almost invariably occurred included micrococci apparently much resembling, if not identical with, the following: (1) *M. pyogenes aureus;* (2) *M. cereus flavus;* (3) *M. albus,* or *M. epidermis;*—in which observation I find my experiments are in agreement with those of Pfeiffer, while doubtless these various micrococci correspond respectively with Buist's so-called orange, yellow and white vaccine. In addition, I occasionally found another organism, which, from its morphological and microscopical characteristics, I believe to be the *Streptococcus pyogenes.* In order to test what action these various microphytes were capable of exerting, they were inoculated, both in pure culture and mixed in various proportions, not only on the calf, but on the human being, and on the rabbit, since this latter animal is apparently susceptible to vaccination performed in the ordinary manner. Such inoculations for the most part produced little or no effect, although inoculation of *Streptococcus pyogenes* alone occasionally caused the appearance of a small circumscribed inflammatory patch around the point at which it was inserted. In one instance also a rabbit died, apparently of septicæmia, after inoculation of a mixed growth of the various organisms previously mentioned, together with a certain amount of broth in which they had been grown. In the whole series of inoculations nothing resembling a vaccine vesicle was seen; so that it was apparently pretty certain that none of the

organisms that had been isolated in the manner described could be directly concerned in the causation of vaccinia.

As it appeared possible that their exuberant growth might be capable of preventing the development of the more important organism for which I was seeking, an attempt was made next to isolate it by some method of treating vaccine lymph which should destroy all extraneous micro-organisms without injuring in any way its potency for purposes of vaccination.

Having in mind the method by which Kitasato succeeded in obtaining pure cultures of the tetanus bacillus, I considered whether it might not be possible in like manner to isolate the specific organism of vaccinia. For the purpose of testing similar measures, I exposed capillary tubes of fresh vaccine lymph to different temperatures for varying lengths of time, afterwards inoculating a portion of the contents of these tubes on the calf, a second portion being used for the making of plate-cultivations in nutrient gelatine and agar-agar. By proceeding in this manner, after numerous experiments, I presently arrived at a temperature at which those organisms, which usually grow so luxuriantly when vaccine lymph is inoculated into nutrient jelly, are apparently incapable of continued existence after exposure to its influence for a certain length of time.

In the first instance I exposed test-tubes, containing half-a-dozen filled capillary tubes apiece, in an incubator at a temperature of 60° C., for an hour at a time, some of them being treated in this manner on one occasion only, while others underwent similar treatment for periods varying from one to six days. At the same time tubes of alkali-albumen solution, specially prepared from blood-serum, were inoculated with vaccine-lymph and intermittently exposed to a similar temperature, in like manner to the different series of capillary tubes.

Plates were then poured from gelatine and agar-agar tubes inoculated both from the capillary tubes and from the tubes of alkali-albumen solution. These plates were in each case poured *direct from the test-tube first inoculated* without the intervention of secondary or tertiary dilutions in the ordinary manner. In no single instance, however, did any growth result, so that it was evident that all the organisms which usually spring up in cultivations of vaccine-lymph had been destroyed.

At the same time a calf was inoculated on certain definitely-circumscribed areas of the abdomen, previously

shaved, with lymph from the capillary tubes, and also with fluid from the alkali-albumen tubes treated in the manner just described, but in this case also the result was negative in every instance, so that evidently the temperature to which the tubes had been exposed had not only destroyed all extraneous organisms, but probably also the true vaccine-virus itself.

Dr. Husband* some years ago found experimentally that exposure of lymph stored in tubes to a temperature of from 80° to 90° Fahr. (33.3° C.) for several hours daily (period not mentioned), did not in any way affect the properties of the lymph so treated; and, on repeating his experiments, I found that such was the case. Obviously, then, it was necessary to seek for a temperature between the limits of 33° C. and 60° C., at which, if possible, extraneous organisms would be incapable of existing without at the same time injuriously affecting the proper action of the lymph.

After a long series of experiments with lymph exposed to various temperatures between the limits mentioned and for varying periods of time, I have apparently determined that the required temperature is one ranging between 38° C. and 42° C. An exposure to the higher of these temperatures for an hour has usually the effect of preventing any growth in plate-cultivations made subsequently, while at the lower temperature a few points of growth are occasionally seen after the lapse of a day or so. At the same time, however, the higher temperature appears occasionally to exert an injurious influence on the lymph as regards the normal vesiculation which should result from the inoculation of vaccine-lymph. Further experiments are therefore needed before the most suitable temperature and most desirable length of exposure can be definitely determined.

When this has been done, provided that the so-called "virus" does in reality owe its peculiar properties to the presence of a living organism, it will be only a matter of time before a cultivation-medium suitable for its growth is found, and a demonstration of its biological and morphological characteristics thus rendered possible.

It does not, however, by any means follow that because no vesicles result from the inoculation of lymph treated in the manner I have described, that no effect is produced on the animal so operated on. Indeed, for a time at any rate,

* Seaton's *Handbook of Vaccination*, p. 153.

there may be protection from the effects of the subsequent inoculation of fresh untreated lymph. This was notably the case in one calf, which was first inoculated, apparently unsuccessfully, with lymph which had been exposed to a temperature of 48° C. for an hour, and a week later inoculated by Dr. Cory directly from another animal, the lymph from which latter animal was at the same time used with perfect success in the case of other calves and of a number of children. On this particular calf, however, the operation performed by Dr. Cory was altogether without result, thus apparently showing that the use of the "treated" lymph, though producing itself no vesiculation, had apparently rendered the animal insusceptible to the subsequent operation with fresh lymph.

Supposing always that a micro-organism is normally present, we may perhaps explain the occurrence of such immunity on the supposition that the temperature to which the lymph had been subjected was sufficient to destroy the vitality of the organism, without, however, affecting the potency of certain of its products in the lymph.

This experiment points to the possibility of obtaining all the desired, without any of the disfiguring, or even absolutely dangerous, effects of vaccination, by the use of such sterilised lymph, either in the ordinary way, or subcutaneously injected after dilution with normal saline solution.

Such a method of procedure would of course render arm-to-arm vaccination an impossibility; but, provided that sufficient lymph could be otherwise obtained, perhaps this would not be altogether an unmixed evil.

Conversely, lymph which has been subjected to the conditions of temperature just described as most suitable for the removal of extraneous organisms, is apparently more potent, in its visible effects at any rate, than lymph freshly obtained. Thus, the calf which Dr. Cory attempted unsuccessfully to inoculate with fresh lymph was at the same time inoculated on another portion of the abdomen with active "treated" lymph, with the result that while, as stated, no visible effect was produced in the large area operated on by Dr. Cory, five vesicles appeared as the result of ten incisions in the area in which the "treated" lymph had been employed.

Early in my experiments my attention was called to the fact that lymph stored in capillary tubes nearly always becomes cloudy after a longer or shorter interval, while at

the same time it often becomes uncertain in its action when subsequently used for vaccination.

I endeavoured, therefore, in the first place to determine the reason for the opacity which occurs in stored lymph, and secondly to find, if possible, some means of preventing such an occurrence. As regards the first point, experiments were carried out as follows. A large number of capillary tubes were filled with calf-lymph, every precaution as to cleanliness and careful sealing of the tubes being observed. These tubes were then set aside, not, as is usually the case, lengthwise, but on end. After the lapse of a few weeks, all the tubes presented little points of opacity, and, on careful examination, it was obvious that each of these occurred where a surface of the lymph was in contact with a bubble of air. Moreover, it was always at the *lower* end of the line of lymph that such opaque points were formed, thus showing that they were composed of something possessing a higher density than the lymph itself, as otherwise there was no apparent reason why they should not also be met with at the upper limit of each thread of lymph where air was also present. This, indeed, was not infrequently the case when the tubes had been left in a horizontal position.

This appearance was quite independent of any coagulation in the lymph; indeed, on breaking some of the tubes in which it was most marked, no coagulum was found. On the other hand, where clotting had taken place after the lymph was stored, the opacity was often not in discrete points, as in the other tubes, but formed with the coagulum a central whitish thread in the midst of a clear fluid. In these cases, the tiny particles of which an opaque point was obviously composed, had apparently become more or less entangled with the fine thread of fibrin which had resulted from the process of coagulation.

Cultivation-experiments were then carried out, gelatine-tubes being inoculated from opaque points in stored tubes, and also as a control from tubes of comparatively fresh lymph, the capillary tubes used in these experiments having been sterilised in each case previous to their being filled with lymph. In each case plate-cultures were made from dilutions of the gelatine-tubes first inoculated. As a result, many more colonies were found in the plates poured from inoculations of the old tubes than from those descended from the fresher lymph. We are therefore apparently justified in considering that the opacity of old-stored lymph

is in the main the outcome of an enormous multiplication of aerobic bacteria, the ancestors of which are present in the lymph when first taken, although the numbers are then so comparatively small as not to render it in any way turbid.

As I have already shown, a certain amount of heat, when applied to lymph in sealed capillary tubes, inhibits the growth of these aerobic bacteria, while a still higher temperature kills them. The exposure of freshly-stored lymph, then, to a proper temperature for a sufficient length of time ought to prevent the subsequent appearance of opacity, and such is indeed the case. Without great care, however, there is in this method considerable danger of rendering the lymph inert. A simpler method of obtaining the desired result, however, is found in the admixture of the lymph with a certain proportion of 50 per cent. glycerine in distilled and sterilised water prior to storage in capillary tubes, which also should have previously been sterilised by heat.

Müller showed long ago that lymph might be diluted with three times its bulk of such a mixture and still retain its properties unimpaired, a fact which has been taken advantage of by more than one purveyor of trade lymph. Experiment shows that not only is this so, at any rate for a considerable time, but also that in tubes filled with such diluted lymph opacity does not apparently result. The glycerine appears to inhibit the growth of those aerobic bacteria which in former parts of this paper I have termed "extraneous", signifying by that term that their presence is not in any way essential—indeed, probably rather the reverse—to the successful action of the vaccine lymph.

Whether or not the actual vaccine virus is capable of increasing in such a proteid and glycerine-containing mixture it is not easy to say; but, before I had read of Müller's advocacy of such treatment, I had employed it, for the reason that other pathogenic organisms have been shown to grow more luxuriantly in a mixture to which glycerine has been added, while with saprophytic organisms this is not the case. Another argument in favour of the use of lymph diluted in this way with glycerine is found in the fact that, as before stated, such a mixture does not dry up nearly as readily as ordinary lymph, and therefore, in the hands of operators all but the most expert, affords greater facilities towards the attainment of

a uniformly successful series of vaccinations. The fact also that a much larger supply of lymph would thus be available is so obvious that it need not be insisted on.

In conclusion, I can only look upon this paper as a preliminary communication; and so, while apologising for the slight amount of progress in our knowledge it records, I bring forward such an account of work that has been done, in the hope that the lines herein laid down may possibly be of service in the further elucidation of this most difficult but most important subject.

ON THE MANIFESTATIONS OF CHOLERA IN SHIPS AT SEA, AND THEIR CONNECTIONS WITH OUTBREAKS ON LAND.

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THE appearance of cholera, in ships at sea, is quite as interesting as its progress over land, and is even of more importance in indicating the factors by which it is diffused. I have been collecting information on this question for thirty years past, and have, on two occasions, placed some of the facts before the Epidemiological Society.* In this I will deal with the connection of the outbreaks in ships, with the indications of the disease on either side of that part of the ocean where they occur.

The first point which presents itself here, for consideration, is the relation between what is generally designated cholera nostras, and the malignant form of the disease common during epidemics. There is evidently a difference between these two, of much the same character as there is between benign plague and the pestilential form of that disease, or even between ordinary intermittent and severe remittent fever; but, epidemiologically, these all resemble each other in this respect, that, when prevailing, there is a central area occupied by the more severe form which is surrounded, in front and on either side, by a zone of the milder disease, often to a great extent. Under such circumstances, a number of cases of the malignant form occur sporadically among the slighter, and, as the edge of the central area is approached, these frequently occur in groups, without being actually in continuation with it. As the epidemic pursues its course, it occupies the portion of this zone in front of it, either at a later period in the same year, or in the following one, while, in those portions on either side of the area of its full development, the cholera nostras and the sporadic malignant cases increase

* Epidemiological Society's *Transactions*, vol. iii, p. 288, and New Series. vol. iv, p. 102.

as the central body of the epidemic passes, and finally cease as it disappears.

Anyone anxious for information on these points, can obtain it by comparing the deaths from cholera in the annual returns of the causes of death in the Reports of the Registrar-General of England and Wales, of Scotland, and of Ireland, for a series of years, in connection with the prevalence of that disease on the adjacent portions of Europe for the same period. The following instances may be here detailed. In 1865 cholera was prevalent in the Mediterranean, and, in the course of the summer, advanced to the northward, through France, reaching Paris on August 24th. Cholera nostras, which had caused 45 deaths per million in England and Wales in 1864, rose to 64 in 1865, and embraced an unusual number of sporadic malignant cases. Dr. Wilkes treated one in Guy's Hospital early in July, and there was another on 27th June in the Borough, which proved fatal on the 28th. In Scotland, where the deaths returned as cholera in 1864 were two in 100,000, these rose to seven in the same number of persons in 1865. And in Denmark and South of Sweden cholerine, accompanied by an unusual number of the malignant form, was much more common in 1865 than for several years previously. Through this extensive area, therefore, indications of the presence and activity of the causes of malignant cholera were more pronounced in 1865 than the previous year, a position accentuated by the outbreaks at Southampton, and Theydon Bois in Essex; though nowhere did the approaching epidemic reach them until 1866, when it invaded Great Britain and the whole of Western Europe up to the Baltic.

The following also may be noticed. In 1837 cholera had been epidemic in part of Italy, Sicily, and at Malta, and had also prevailed in Russia and Germany. In August, four cases and two deaths occurred in the *Dreadnought* hospital-ship in the Thames; and in October, between the 8th and 28th, there was another outbreak in her, during which twenty were attacked, of whom twelve died, when the disease ceased. In 1837, also, there was a sporadic case near Edinburgh in September, which recovered; another in November, and a third in December, both of which proved fatal. There was a severe outbreak in the House of Industry at Coventry between 7th January and 5th February 1838, in which there were fifty deaths from cholera.

In 1859, when cholera was epidemic along the western

coast of Europe, from South of Spain to Sweden, there was a limited outbreak at the coastguard-station on Southampton Water, near Netley, between 3rd and 17th July. In the Marshalsea prison at Manchester, the prisoners were attacked by choleraic diarrhœa on 28th September, and, out of the 466 in confinement, 196 (including both males and females) were reported sick on 28th, 56 on 29th, 9 on 30th, 5 on 1st October, and 2 on 2nd—256 in all. The water was supplied from the Salford Waterworks, and was stored in the gaol in two cisterns—one attached to the kitchen for the preparation of food for the prisoners, the other for the officers of the establishment. The weather was very hot at the time, and the water in the first named, on the day of the outbreak, was found of a yellow colour and insipid in flavour, and, on examination, the overflow-pipe, which opened into one of the sewers, was found untrapped. The water in the other cistern was found clean and refreshing, and none of the fifty-three officers and others on the premises, who did not employ the water from the kitchen cistern, were affected. In addition to these instances, twelve persons died of cholera, between 1st and 17th October, at Glass Houghton, a village near Pontefract in Yorkshire. There was also a sharp outbreak of cholera in the end of summer at Wick, in the North of Scotland. The deaths from cholera in England, which were 34 per million living in 1858, rose to 45 in 1859, and in 1860 they fell to 14; in Scotland the ratios per 100,000 living in the corresponding years were 3, 5, and 2, showing the same fluctuation, and practically to the same extent. Had space permitted, a great many more instances of a similar description could have been given; but those detailed above are sufficient to show that the cholera nostras and the sporadic cases in the malignant form, surrounding the area occupied by an epidemic of cholera, are under the influence of the factors causing that epidemic, and form part of the natural history of the disease, which cannot be overlooked when its mode of diffusion comes to be investigated.

A very important observation, with reference to cholera, was made by the late Dr. Prout sixty years ago, which epidemiologists of the present day seem to have altogether overlooked. Dr. Prout had been engaged, in London, for about six weeks, in ascertaining the weight of a given bulk of atmospheric air, under the same conditions as to temperature and pressure, when, on 9th February 1832, he found it had acquired a small, though sensible, increase. This,

though not quite so great as on the first day, continued while the observations were made, which was for six weeks longer. About the 9th February the wind, which had previously been westerly, veered round to easterly, and remained chiefly in that quarter till the end of the month. Now, precisely on the change of wind the first cases of epidemic cholera were reported in London, and from that time the disease continued to spread. Dr. Prout ascribed the increased weight to the admixture of some form of malaria, heavier than air, but, as the matter then appeared to him, he thought it more perhaps than could be safely maintained that the cholera was due to this foreign matter, though he states his impression that epidemics of that disease are due to such a cause.

Dr. Bryden, in his *Report on the General Aspects of Epidemic Cholera in* 1869, published as a sequel to his *Report on the Cholera of* 1866-68, gives (at p. 54) the following details with reference to the coolies who proceeded from Calcutta to Mauritius in 1850-60, and to the West Indies in 1861-69. For Mauritius 105,382 embarked, of whom 22,077 in ships that became affected with cholera, and of these 288 died of it; thus 21 per cent. of the total number embarked were exposed to the disease, and 13.0 per 1,000 of those so exposed died of it. For the West Indies 72,681 embarked, 8,284 of them in ships that subsequently became affected with cholera, and 89 died of it; 11.4 per cent of those embarked were therefore exposed to cholera, of whom 10.7 per 1,000 died of it.

The Sanitary Commissioner with the Government of India, in his Report for 1881, has given (at p. 135) a statement showing that 222 vessels, carrying 129,527 coolies, left Calcutta for Mauritius, Natal, and the West Indies from 1871 to 1880. Cholera appeared in thirty-two of these, with 14,572 coolies on board, being 14.3 per cent. of the total, and of these 181, or 12.3 per 1,000, died of that disease. Though the percentage of ships attacked in the first period was nearly double of that in the following two, the mortality per 1,000 exposed was nearly the same in all, showing the incidence of the disease to have been very nearly the same in each period.

In the statement for 1871-80 the deaths from cholera each day after embarkation are given for each ship up to the twentieth, and from the twenty-first onwards in one group. The first death did not occur on fifth or sixth day after embarking, nor on the seventeenth, eighteenth, nineteenth, or twentieth days. There were thus three

periods of attack, the first extending from the first to the fourth day, the second from the seventh to the sixteenth day, and the third from the twenty-first onwards. In the Table, page 86, are given the names of the thirty-two vessels, the number of coolies embarked in each, with the date, the deaths from cholera in each of the three periods, with their total in each ship. Also the dates of landing and the points of disembarkation.

Sailing ships, proceeding from Calcutta to the southward of the equator, go along the meridian of 90 deg. E. as nearly as they can, so as to cross the line from that to 93 deg. or 94 deg. E. After passing through the variable winds met with between the equator and 4 deg. to 12 deg. S. (according to season), they reach the S.E. trade, which is a fair wind for those going westward, but those for the east have to pass through the trade, to avail themselves of the westerly winds which prevail to the south of it.

After embarking their emigrants, the sailing ships—since 1871, at least—seem to have been towed out of the river, so as to be in the open sea on the second day; in estimating their position, subsequently, no material error will be committed if the seventh, sixteenth, and twenty-first days after embarkation be reckoned as the sixth, fifteenth, and twentieth days after leaving the land. Employing the data in my possession, the approximate latitude of sailing ships from Calcutta, on the sixth day at sea, is about 15 deg. N.; on the fifteenth day about 7 deg. N.; and on the twentieth day about 1 deg. N., with an extreme variation in individual cases of about 3 deg. either way. It thus appears that, in sailing vessels, the first of the three outbreaks noticed above must have occurred near the 90 deg. meridian, and to the north of about 16 deg. N.; the second from 15 deg. N. to 7 deg. N.; and the third about 1 deg. N. onwards; and the factors which determine them must have become active about these positions respectively. The deaths in the first group, being within the usual period of incubation, may be due to exposure on shore; but those in the second, and still more in the third groups, cannot be explained in this way.

Referring to the Table,* now it is seen that from 11th September 1871 to 3rd November 1874, a period of three consecutive years, twenty-five ships were attacked with cholera, while from 1875 to 1878 inclusive, the four

* This Table is a re-arrangement of the facts contained in that at p. 114 of vol. iv of the Epidemiological *Transactions*, New Series, to bring out relations with the land these do not deal with.

TABLE I.

Abstract of Coolie Ships which sailed from Calcutta during 1871-80 *in which Cholera appeared.*

Ships' Names.	Numbers embarked.	Date of Embarkation.	Deaths from Cholera.			Total.	Date of Disembarkation.	Destination.
			1st Period.	2nd Period.	3rd Period.			
Adamant	343	Sept. 22, 1871	—	1	1	2	Dec. 23, 1871	Demerara.
Medea	431	Nov. 8, „	—	2	9	11	Jan. 26, 1872	„
Poonah	506	Feb. 22, 1872	—	13	5	18	June 18, 1872	„
Fateh Salam ...	275	Mar. 7, „	5	—	—	5	Apr. 13, „	Mauritius.
Humber	462	Mar. 31, „	—	—	1	1	Sept. 9, „	Jamaica.
Wellesley	334	May 14, „	—	1	—	1	June 26, „	Mauritius.
Woodburn ...	577	Aug. 1, „	—	5	—	5	Nov. 3, „	Trinidad.
Kate Killock ...	456	Aug. 5, „	—	14	—	14	Nov. 12, „	Demerara.
Sea Queen ...	328	Sept. 4, „	1	1	—	2	Dec. 2, „	Jamaica.
Neva	484	Nov. 23, „	1	3	—	4	Feb. 25, 1873	Demerara.
Shah Jehan ...	285	Mar. 30, 1873	—	—	2	2	May 8, 1873	Mauritius.
Allumghier ...	340	May 28, „	—	1	—	1	June 30, „	„
Enmore (steamer)	577	July 4, „	2	3	5	10	Aug. 25, „	Demerara.
Merchantman ...	383	July 25, „	—	4	—	4	Sept. 1, „	Mauritius.
Syria	438	Aug. 24, „	—	4	2	6	Nov. 27, „	Trinidad.
Golden Fleece ...	524	Sept. 2, „	—	—	6	6	Nov. 30, „	Demerara.
Sir Henry Lawrence.	462	Sept. 16, „	1	5	6	12	Dec. 15, „	„
Hereford	588	Sept. 20, „	—	—	3	3	Dec. 9, „	„
Loch Lomond ...	518	Jan. 27, 1874	—	11	—	11	Apr. 21, 1874	Jamaica.
Rohilla	425	Feb. 17, „	4	—	—	4	May 11, „	Demerara.
Blenheim (steamer).	692	June 13, „	—	—	5	5	July 8, „	Natal.
Golden Fleece ...	519	Aug. 6, „	—	—	2	2	Oct. 29, „	Trinidad.
Forfarshire ...	509	Aug. 18, „	—	13	1	14	Nov. 5, „	Demerara.
Lincelles	342	Oct. 7, „	—	7	—	7	Jan. 8, 1875	St. Vincent.
British Empire	633	Nov. 8, „	—	—	2	2	Feb. 25, „	Trinidad.
Lady Melville ...	391	June 12, 1875	—	—	1	1	July 24, 1875	Mauritius.
Atalanta	393	Mar. 22, 1876	1	—	—	1	May 13, 1876	Natal.
Linguist	600	Aug. 2, „	—	1	—	1	Oct. 20, „	Demerara.
Artist	552	Sept. 30, 1877	2	6	—	8	Dec. 29, 1877	„
Boyne	568	Feb. 13, 1878	—	7	6	13	May 15, 1878	„
Glenroy	486	May 18, „	1	—	—	1	July 17, „	Natal.
Ophir	351	June 19, „	—	4	—	4	Aug. 22, „	„
None	—	1879 and 1880	—	—	—	—	—	—

years following, there were only seven attacked, and in the next two, 1879 and 1880, there were none. From 1871 to 1874 six ships showed cholera in the first period, two of these had no death subsequently; two others, which had had one death early in the first period, had one, and three, respectively, in the second, and no more; and the other had deaths in all three periods. Of the remainder, twelve had their first death in the second period, of which five had others in the third; and there were seven attacked in the third period. From 1875 there were three ships in which cholera appeared in the first period, and in one of them again in the second; and in three others in the second period, and in one of them again in the third period; there was but one instance, the *Lady Melville*, in which the first death occurred in this.

The positions of the different ships named in the Table may be made out, approximately, to the twentieth day, from the remarks already given, but, as their longitudes are not mentioned, there will be more uncertainty after this, though they will not deviate materially from the track of those on the accompanying map. The mean duration of the voyages of the sailing vessels, from Calcutta to Mauritius, was forty days, and to Natal sixty days. I have records of two ships—the *Gertrude*, which left Calcutta in May 1859 with military invalids, which had eight attacks of cholera, the first on the seventeenth day out, in 2 deg. N., 89 deg. E., and the last on the thirty-fourth day out in 24 deg. S., 58 deg. E., just south of Mauritius; and the *Alnwick Castle*, in November 1861, with coolies, one of whom was attacked with cholera on the thirtieth day out, in 23 deg. S., 57 deg. E., also south of Mauritius. The steamer *Blenheim* landed her coolies at Natal on the twenty-fifth day after embarkation; being a full-powered vessel, she would make a straight course for Ceylon and Mauritius, and from that to Natal. As the distance from the Hooghly to Mauritius, by this track, is 3,180 miles, and from there to Natal 1,500, assuming the ship to have made equal progress daily (and it would not have differed materially from this), she would have been off Mauritius in seventeen days, so that the outbreak commenced after she had passed that island. The *Enmore*, another steamer, which landed her emigrants at Demerara on the fifty-second day after embarkation, would have pursued the same course; she must have been south of Mauritius about the seventeenth day after sailing. At Mauritius itself there were a considerable number of

sporadic cases at the end of 1870, and in the course of 1871, and early that year, cholera became epidemic at the Seychelles Islands, 900 miles north of Mauritius, and along the east coast of Madagascar; but I have no record of it having been met with between 1871 and 1880, either at Natal or in the Cape Colony. On the other hand, it is to be remarked that, from 1868 to 1871, an epidemic of cholera, which was first heard of in latitude 5 deg. N. and longitude 38 deg. E., in the former year, passed southwards, reaching Zanzibar at the end of December 1869, and Quillimane at the mouth of the Zambesi in 1871. This was met with to the west of Lake Tanganyika and Lake Moero, upwards of 700 miles from the coast, and under it there were outbreaks in Johanna and Mayotta, two of the Comoro Islands, and at Majunga on the west coast of Madagascar, and at Nossi Be, a small island north of that place.

While the changes in the distribution of cholera, detailed above, were in progress, a very striking reduction in its incidence in British India was also taking place, as will be obvious from the numbers in Table II of the deaths registered in the various provinces during the years 1869

TABLE II.

Districts.	1869.	1870.	1871.	1872.	1873.	1874.
Punjaub...	9,258	469	169	8,727	148	78
N.W. Provinces and Oude.	92,676	45,451	19,505	77,131	19,229	6,464
Bengal	—	6,511	20,396	46,901	64,366	56,876
Central Provinces and Berar. Bombay Presidency Madras Presidency	135,728	59,517	24,111	32,059	1,467	366
British Burmah ...	1,025	250	162	640	8,109	960
Ceylon	5	301	61	Not known	14	11

Districts.	1875.	1876.	1877.	1878.	1879.	1880.
Punjaub...	6,246	5,736	29	215	26,135	274
N.W. Provinces and Oude.	64,427	48,311	31,770	22,221	35,892	71,546
Bengal	112,276	196,590	155,305	95,192	136,363	39,643
Central Provinces and Berar. Bombay Presidency Madras Presidency	179,410	203,113	418,918	169,201	48,031	1,628
British Burmah ...	761	3,678	7,276	6,759	1,828	2,638
Ceylon	1,817	Not known.		Not known.		799

to 1880 inclusive. The registration for Bengal was so defective in 1869 that the results were not published; that for 1870 in the Table is for the last six months of the year, and that for 1871 is evidently too low.

The numbers in this Table show a great reduction from 1869 to 1871 (Bengal excepted). In 1872 there was an increase in all the Provinces, very considerable in Bengal and that to the north of it, but to a much smaller amount in those to the south. In 1873 there was again a marked diminution in the number of deaths in all the Provinces except Bengal and Burmah; the reduction in the North-West Provinces was to one-fourth of those in 1872, and in the Central Provinces, Berar, and the Bombay and Madras Presidencies, to a twenty-third of the deaths in 1872; in 1874 there was a still further decrease, reducing the numbers in the North-West Provinces to one-twelfth of their amount in 1872; and in the Central Provinces, Berar, and Bombay and Madras Presidencies, to one eighty-eighth. In the latter year, indeed, cholera had almost disappeared from these extensive areas. In 1875 there was an enormous increase of the mortality of cholera all over Hindostan, from Cape Comorin to Lahore.

While these occurrences were in progress, cholera was extremely prevalent in the countries to the eastward of the Bay of Bengal and Indian Ocean. Cornish, in his *Sanitary Report for Madras*, for 1873, states, at p. 91: "While the Peninsula of India has on this occasion escaped invasion, it must not be forgotten that cholera has been noticed in great force in the territories to the east of the Bay of Bengal. In Burmah, Tenasserim Provinces, Cochin China, the Straits Settlements, and the Islands of the Indian Archipelago, the destroyer has reaped a goodly harvest of victims."

The deaths from cholera in the Burmese territory, in the Table, show there were considerable fluctuations in different years, varying from 162 in 1871, to 8,109 in 1873; declining again to 761 in 1875. The Lisbon authorities declared the Island of Timor affected as early as February 1872. British Burmah, consists of a narrow slip of territory running southward along the eastern side of the Bay of Bengal from Chittagong to Cape Negrais, in lat. 16 deg. N. (which is separated from the Valley of the Irrawaddy by a high range of mountains), together with the valleys of the Irrawaddy and Saltang River to lat. 19 deg. N., and a narrow portion along the western shore of the Peninsula as far as 10 deg. N. The behaviour of cholera in these two

divisions differs materially, both as to time of appearance and to severity, as will be obvious from the following, in which the deaths in the above Table, for the whole district, have been distributed to the sub-divisions in which they occurred.

Years.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.
In Northern Sub-division.	560	11	80	212	1308	2357	1518	444	126
In Southern Sub-division.	80	8098	880	548	1424	4919	5241	1384	2512

These figures show that the northern sub-division, so far as cholera is concerned, is epidemiologically connected with Bengal, while the southern sub-division is more intimately connected with Siam, Cochin China, and the Archipelago to the south and east of it. There is no clear account of the commencement and increase of the epidemic in this extensive area, but the announcement of the Portuguese Board of Health, that cholera was at Timor in 1872, and Mr. Cornish's description of it in 1873, with the deaths in Burmah in 1873 and 1874, leave but little doubt on its general features and progress.

It has been shown above that sailing-ships from Calcutta, on the sixth day after leaving the land, will have reached 15 deg. N. on the 90th meridian; and by the twentieth day will have got to about 1 deg. N., near the same meridian. From the sixth day out, therefore, they will be to the south of Cape Negrais, and any emanations carried to the south-westward from the Valley of the Irrawaddy, and the cholera districts to the east and south of it, will pass directly over their track in the second period in the Table given above, viz., from the sixth to the twentieth days at sea; after the twentieth day, when they have got into the third period, they will still be exposed to the same influence; but as there are no longitudes given for the sailing-vessels in the Table, the extent to which it is operative in these instances cannot be indicated, but others will be noticed hereafter in which the disease occurred as far as Mauritius, and even beyond it. The *Blenheim*, however, a steamer referred to above, from Calcutta to Natal in 1874, was on the fourth day out in lat. 10 deg. N., long. 83 deg. E.; on tenth day 4 deg. S., 73 deg. E.; and on seventeenth day 20 deg. S., 57 deg. E., close to Mauritius. There was no death from cholera in the *Blenheim* until she passed Mauritius. The

Enmore, also a steamer, for Demerara in 1873, which would have passed over the same course, and at much the same rate as the *Blenheim*, had two deaths by the fourth day out, one on the tenth, one on the seventeenth, and one on the nineteenth, besides five after the twentieth, must have had the case on the seventeenth while she was off Mauritius, and the other six after passing that island.

This great reduction in the activity of cholera at sea, in 1875, was followed by its return in force over those portions of Hindostan from which it had almost disappeared in 1873 and 1874, and the steps by which its return was indicated are worthy of examination. For many years now the general belief has been that the cause of cholera becomes active in the endemic area in Lower Bengal, and spreads from that locality not only over Hindostan, but beyond its boundaries into other countries; in conformity with this idea it was concluded that Ceylon always received its epidemic of the disease from the adjacent part of the Madras coast; different epidemiologists attributing the diffusion, some to aerial currents, others to communication from man to man, while pursuing their ordinary avocations.

Taking the deaths from cholera in the first four months of 1875, for the Provinces already referred to, they stand as follows:

TABLE III.

Districts.	January.	February.	March.	April.
Punjaub	4	4	4	10
N.W. Provinces and Oude	35	216	1,923	14,757
Bengal	5,530	5,941	11,821	12,954
Central Provinces and Berar ... ... / Bombay Presidency / Madras Presidency	9	24	109	956
British Burmah	1	—	8	25
Ceylon	16	70	281	102

Here Bengal, with a population of sixty millions under registration, shows an active epidemic in progress from January onwards; the North-West Provinces and Oudh, with fifty-six millions under observation, present an increase from January, at first slight, but more marked in March and April, though this was almost confined, at this time, to that portion of them lying between Bengal and Oudh. In the Central Provinces, Berar, the Madras and

Bombay Presidencies, with a population of fifty-six millions, occupying most of the area from Bengal southwards to Cape Comorin, the deaths from cholera to March, inclusive, were 142 only, while in Ceylon, where the population was only about two-and-a-half millions, 367 died from this disease, showing a much greater activity of its causes in Ceylon at that time than any part of Hindostan to the south of Bengal. Since the registration of deaths was introduced in India, it has been found that, in addition to the endemic area in Lower Bengal, there are other localities where, for years in succession, a month seldom passes without one or more sporadic cases, and, in 1875, as the season advanced, it was in these localities where the earliest cases occurred; and the disease radiated from them, gradually occupying the intervening country. There are two such localities on the east coast of the Madras Presidency, to the south of Madras, South Accot, and Tanjore, the latter being the delta of the Cauvery, in about 11 deg. latitude N.; the deaths from cholera in these, and in Trichinopoly, which lies between them, in the first four months in 1875, were as follows:—

	January.	February.	March.	April.
South Accot	3	6	—	3
Trichinopoly	—	—	1	—
Tanjore	4	7	2	65

Amounting to ninety-one in a population of close on five millions, while in Ceylon, during the same period, there were 469 among a population of half the number. The epidemic seems to have commenced in Colombo and its neighbourhood early in January, and to the end of April was confined to the southern half of the west coast, from Negombo to Galle, Negombo being 240 miles from the nearest point of the Tanjore territory in Madras. It is obvious, therefore, that Ceylon must have been subjected to the action of the cholerific factors quite as early as the Tanjore locality, and in a more concentrated form, and as the places first affected in Ceylon were in the southern part of the West Coast, instead of the northern part, the weight of the evidence is altogether opposed to the conclusion that they were carried by man.

In Table I the *Lady Melville* is shown to have had one death, after the twentieth day at sea, in 1875; and, in 1876, the others had single deaths, viz., the *Atalanta*, on

the third day after embarkation, and hence referable to the shore, as being within the incubation period; the other in the *Linguist*, on the sixteenth day after embarkation. In 1877 and 1878 four ships were attacked, which presented twenty-six deaths from cholera, distributed over the three periods in the Table; of these, the *Glenroy* had a single death on the third day after embarkation; the *Artist* had two by the fifth day, and six more by the twentieth; the *Ophir* had four from the fourteenth to the nineteenth; and lastly, the *Boyne* had seven from the tenth to the twentieth day after embarkation, and six more after the twentieth. In Bengal cholera had been prevalent from 1876 to 1879, but in 1880 there was a large reduction. In the North-West Provinces and Oudh the deaths had fallen from 64,427 in 1875 to 22,221 in 1878, from which they then increased to 71,546 in 1880. In the Central Provinces, Berar, and the Madras and Bombay Presidencies, in 1877, owing to the severe epidemic of cholera, combined with the famine in the Madras Presidency, the deaths rose to 418,918, from which they fell, by large reductions, to 1,628 only in 1880. The deaths from cholera in Burmah, in 1877, were almost ten times more numerous than in 1875, and continued nearly as frequent in 1878; in both years far the greater portion of them were in the southern division of that country, as had been the case in 1873 and 1874. I have very little information about the prevalence of the disease in the countries to the east of Burmah in 1877 and 1878, but the French transport *Corrèze*, which left Saigon in July of the former year, with 500 troops and other passengers, for Europe, was attacked a few days after sailing, for which she was afterwards sent to Gebel-el-Tor, near Suez, under observation, though ultimately permitted to pass through the canal in quarantine. The occurrence of these few cases of cholera, with a marked increase of its frequency in the southern division of the Burmese territory, notwithstanding the enormous difference in the incidence of the disease over the southern portion of the Indian Peninsula, merits attention.

If, now, the circumstances under which epidemics of cholera reached Mauritius formerly be examined, it will be found that unusual frequency of the disease along the southern portion of the eastern coast of the Bay of Bengal and among the territories and islands to the eastward had preceded that extension. In 1819 cholera was epidemic in Ceylon, also along the southern portion of the Burmese

territory already referred to, along the peninsula of Malacca, including the town so named, at Bangkok in the kingdom of Siam, at Acheen in Sumatra, and at Java. The *Topaze* frigate arrived at Trincomalee, in Ceylon, on 5th September, with a large sick-list, mostly dysentery. At Trincomalee one man died of cholera on 16th September, two others on 20th, and there seem to have been several recoveries. She sailed for Mauritius on 9th October, and soon after cholera broke out again, and there were seventeen attacks, of which four proved fatal on board. The *Topaze* arrived at Mauritius on 19th October. Thirty-six men were sent on shore to the military hospital, where six of them died, five from the sequelæ of cholera. No case of cholera occurred on the *Topaze* after her arrival at Mauritius, but in three weeks it commenced among the population. It is to be observed here, as having a most important bearing on the question of the introduction of cholera to Mauritius on this occasion, that a Board, assembled by Major-General Darling, the Governor, on 23rd November 1819, to investigate the point, stated, in their report, "that the first well-marked case of the present disease occurred on the 6th September last, and was treated by Mr. Trebuchet in Port Louis" (the *Topaze* did not reach the island until the 29th of October); "it differed nothing from the cases which have presented themselves since the 18th and 19th instants."* The troops at Mauritius had a ratio of 103 attacked and 16.6 deaths per 1,000 from cholera in 1819, and of 132 per 1,000 attacked, with 9.8 deaths in 1820. At Cape Town there was a ratio of 1.7 attacks per 1,000 in 1819, and of 4.1 attacks in 1820, without any death. The disease also prevailed on the east coast of Madagascar, and slightly in the Island of Bourbon.

The next well-marked epidemic of cholera in Mauritius occurred in 1854. Cholera had been prevalent, in 1853, in Rangoon and the country to the south of it, as also in Sarawak and Borneo; but I have no record of its frequency in the intermediate countries. In 1854 the *Sultany* sailed from Calcutta for Mauritius on February 10th, with a crew of 80 men and 375 emigrants; on 28th February, when she had been a fortnight at sea, cholera broke out. She arrived at Mauritius on 25th March, thirty of the passengers having died of cholera during the voyage; some more occurred during her stay in the roadstead of Port Louis before the emigrants were landed at the lazaret.

* *A Treatise on Asiatic Cholera*, by C. Macnamara, London, 1870, p. 41.

There they continued healthy until they were liberated, on 1st May. The crew of the vessel had remained healthy all the time. A Board of seven members, five of whom were experienced resident physicians, appointed by the Governor to examine into the history of the epidemic, reported, among other matters, that the earliest cases in Port Louis occurred on 14th May, among the inmates of the prison, which at the time was much crowded and badly drained. They also reported that five cases of cholera had occurred in the town between the beginning of the year and the arrival of the *Sultany*. As showing that the epidemic influence was active about this time far to the south-west of Mauritius, it is worthy of notice that about April 1853 an hospital-attendant of the 43rd Regiment, at King William's Town, in the Cape Colony, had a well-marked attack of cholera.

In 1856 there was a fresh visitation of cholera, commencing in March, and continuing till the middle of May; the deaths in the colony from it amounting to 3,303. A Special Committee of the Legislative Council (among whom was no medical man) was appointed by the Governor to inquire and report on the cause of this outbreak, and whether it was expedient to make any change in the quarantine laws then in force. Notwitstanding the general doubts expressed by the leading medical men of the colony, the Committee, with one dissentient, did not hesitate to ascribe the epidemic directly to importation by a steamer at the quarantine-station, on board which one of the crew had sickened, although no communication could be traced between this man and the earliest attack in Port Louis, and none of the officials or police of the lazaret were affected. The result was, that still more stringent quarantine regulations were adopted in the next session of the Legislature.

In 1857, and still more 1858, cholera became active at Singapore, Hong Kong, in the Philippine Islands, and along the coast of China. The *Gertrude* left Calcutta on 21st May 1859, with 120 military invalids on board, and some other passengers and her crew, for England, and on 27th got to sea; some cases of dysentery and diarrhœa seem to have occurred after sailing. Cholera appeared on June 12th, in about 2 deg. N. and 89 deg. E.; and dropping cases occurred, the last on 29th June, about 24½ deg. S. and 58 deg. E.; altogether there were nine cases of cholera, of which eight proved fatal.

The *Alnwick Castle* from Calcutta, with coolies, had a

single case of cholera on 27th November 1861, in 23 deg. S., 57 deg. E., just south of Mauritius. She had been thirty days at sea, having had no communication with land or any other vessel.

The military returns show slight indications of cholera among the troops in Mauritius in 1859 and 1860, and it appeared in an epidemic form among the civil population on two sugar-estates, on the elevated ground of Plaines Wilhelms, between six and seven miles from Port Louis, in September; the cause was not detected. After a lull from the 25th November to 13th December it reappeared, and ceased at the end of 1860. In December 1861 cholera became epidemic again in Mauritius, and continued till the middle of 1862, and it is said caused upwards of 4,000 deaths. There were traces of ordinary cholera at Cape Town in 1859; and in January or February 1861 a case of malignant cholera, which passed into collapse, but recovered, and much choleraic diarrhœa at Maritzburg in Natal.

In 1863 and 1864 there was again much cholera met with in Burmah, Singapore, Java, in the Philippine Islands, and at Hong Kong, and it was still active in the last-named in 1866. A ship, the *Queen of the North*, left Bombay for England on 22nd January 1864, with a crew of twenty-seven, and 272 passengers consisting of officers, time-expired men, some invalids, women and children. Several of the invalids and women and children had suffered from bowel-complaint previous to embarkation. One woman, whilst under treatment for diarrhœa, was attacked on the evening of the 24th with vomiting and purging, with a disposition to collapse, and died the following evening; but the medical officer hesitated to call the case cholera. Two other cases with collapse, but without cramps, and with bilious stools, occurred on the 28th and 29th January; both recovered. The first unequivocable case of cholera appeared on 1st February, and proved fatal the same day; the ship's position on this day was 2 deg. 49 min. N., 77 deg. 7 min. E, with a fine north-easterly breeze. From this to the 7th there were slight breezes and calms, and a heavy thunderstorm on the latter, and three cases of cholera occurred. Slight winds and calms prevailed until the evening of the 13th, when the S.E. trade wind commenced about 10 deg. S., and by the 15th the ship's position was 14 deg. 3 min. S., 77 deg. 18 min. E. From the 8th to the 15th, inclusive, there were thirty-seven attacks of malignant

cholera, of which twenty-four proved fatal. There was no attack after the 15th, and the last death was on the 19th. The provisions were good, but the water, which had been obtained at Bombay, always became opaline after exposure to the air for a little. Diarrhœa was very common, among both passengers and crew, from about 5th February; the crew who were berthed in the forecastle, which had no communication with the hold, had no cholera; but the chief officer, who messed in the cabin, and drank large quantities of water, was attacked and died. The cases of either disease did not come from any particular part of the troop-deck, but were spread all over it. It will be seen by the ship's positions, when the violent outbreak commenced on the 8th February, that, though coming from Bombay, she was then not far from the common track of sailing-vessels from Calcutta after they crossed the line. That the water had something to do with the sudden outbreak of cholera seems highly probable, but its abrupt termination, notwithstanding the same water continued to be employed, would indicate that there was something connected with the locality, for the time being, of the greatest importance in its production. Another ship, the *Durham*, embarked, on the 24th February 1866, thirteen officers, 422 non-commissioned officers and men, and forty women and children, for England, and seems to have sailed the following day. Ten cases of cholera occurred during the voyage, the first on the 9th March, when she was in 10 deg. 15 min. N., the next on 19th March, when she was just south of the line, and the remainder onwards, the last on the 1st April, when she was 20 deg. 0 min. S., and 72 deg. 10 min. E. There were five deaths in all from cholera. The *Durham* at this time was close to the position of the *Queen of the North* on the 15th February 1864, though, fortunately for the passengers, her experience was so different. In September 1865 there were several coolie ships in quarantine for cholera at Mauritius, and sporadic cases on shore. At Cape Town, in February 1865, there were many cases of severe cholerine; and at Port Elizabeth, one case of severe common cholera in end of October 1865; and on 12th November, at sea, in 33 deg. S., 37 deg. E., one case, a soldier of the 96th Regiment, on board the *Renown*, which had been in quarantine since her arrival from Gibraltar on 9th October, and had been thoroughly disinfected before the 96th embarked on 9th November at Port Elizabeth. In October 1866 a lady in Cape Town, who had not been out of the colony, died of

malignant cholera. At Maritzburg, in Natal, cholerine, diarrhœa with severe cramps in abdomen and limbs, with tendency to collapse, were frequent in end of December. The *Alumbagh*, from Mauritius to Port Elizabeth, had a single case of common cholera about 1st March, when midway between these two places. The *Windsor Castle*, with troops on board for Kurrachee (mentioned farther on), which had been affected with cholera on her passage from England in 1866, and had the last seizure in the Atlantic, in a soldier's wife, on 20th August, which terminated fatally on September 3rd, in 34 deg. S., 8 deg. E., from secondary fever, had a seaman attacked on September 15th (after she had passed the Cape), in 37 deg. S., 32 deg. E., who died in a few hours.

The above details place it beyond doubt that ships traversing the Indian Ocean pass through localities in which their passengers suffer from cholera many days after they have left the land and its immediate influences; and as, in most instances, there has been no succession of cases to keep up the disease from the date of sailing, its occurrence must be attributed to factors they encountered in the localities where they suffered. These factors occupy fresh localities from time to time, as they do on land; and when that from Mauritius to the east coast of Africa is involved, as shown by the appearance of cholera on shore, ships passing in the vicinity of these points have had one or more cases, though they had been many days at sea, and without any recent or even any previous case, the essential factors must then be altogether independent of the shore or of previous cases, and, in fact, can only be those designated epidemic or pandemic causes.

A few remarks here on epidemics of cholera which have prevailed on the south-east coast of Africa, with their modes of invasion, will prove useful in elucidating this question, and placing its relations to those traced above in a clearer light than it stands at present. We are chiefly indebted to Dr. Christie, who was physician to the Sultan of Zanzibar (now of Glasgow), who, in his work on *Cholera Epidemics in East Africa*, published in 1876, detailed the various epidemics which had shown themselves at Zanzibar and its neighbourhood, and described the caravan-routes from the coast to the interior followed by the native traders in the pursuit of their ordinary traffic. Dr. Christie mentions the epidemics of 1821, 1835, 1858-9, 1865, and of 1869-70. Of the first, that of 1821, no satisfactory information could be obtained at Zanzibar; that of 1835 seems

to have been an extension south-west of an outbreak in southern Abyssinia in 1834, mentioned by Dr. Kirk; it reached Zanzibar about November 1835, and passed on to the southward, but how far is not known. The epidemic of 1858-9 was thought by Dr. Christie to be an extension of that which was in Abyssinia in 1856. The next invasion is supposed to have started from Berbera. Dr. Christie remarks it as first heard of at Merka, a port 2 deg. N., in a severe form, but it broke out at Lamoo, 2 deg. S., and reached Zanzibar at the end of November. It subsequently proceeded south as far as Cape Delgado, causing great mortality. It does not appear to have gone far inland. The epidemic of 1865 passed from Berbera, opposite Aden, south along the caravan-route as far as Gannah and Barderah, on the river Jub, and thence to the coast; it reached Melinda in June, and was experienced a little further south at Mombassa, where the south-west monsoon set in strongly, by which its southerly progress was stopped and its course turned in the opposite direction, and more over the land. The epidemic did not reach Zanzibar. The last outbreak was in 1869-70. Dr. Christie attributes the origin of this to that of 1866, in Abyssinia; it was first heard of by the native traders as being in a locality about 5 deg. N. and 37 deg. E., about the end of 1868. If from Abyssinia, it must have been dormant for some time, as the interval is very large for the space it would have covered had it been in motion. This epidemic, in the first nine months of 1869, gradually overspread the country from 1 deg. N. to nearly 6 deg. S., between the meridians 34 deg. and 39 deg. E. It reached the coast-town Pangani in October, and Zanzibar a few days later, and subsequently affected the Portuguese settlements at Mozambique, and, in 1871, extended to Quillimane, and, inland, prevailed on the west side of Lake Tanganyika.

Proceeding now to the Atlantic, though there are many instances in which ships carrying emigrants from Europe to America have suffered severely from cholera, still the records of most are so deficient in details, necessary to admit of a satisfactory analysis being made of the various factors which have contributed to that result, that they are of little use in this inquiry. Of those which are available for the investigation, the instances of the *Swanton* and *New York*, ships which carried 280 and 338 emigrants from Havre, in the end of 1848, to New Orleans and New York, respectively, are most valuable. The emigrants were Germans, and had passed about two months at Havre

in a wretched condition, while waiting for embarkation; there was no cholera at Havre during their stay, but one report stated some few had arrived, from infected places in Germany, in time to embark with the others. The *Swanton* sailed on 31st October 1848, and the *New York* on 9th November. On the 23rd and 24th the *New York* had a very cold wind from the north-west, followed by a very warm one from south-east on 25th, and on this day, in lat. 42 deg. N., long. 61 deg. W., the first case of cholera occurred. The *Swanton* had no cold wind on 24th, but on the 25th, as with the other, the wind became south-east, and was described by the captain as unlike any he ever experienced before, and was more like artificially heated air than anything else. The first case of cholera appeared in the *Swanton* the following day, the 26th November, when in lat. 26 deg. N., long. 57 deg. W., she being then about 1,000 miles south of the *New York*. On the 24th there was a general opening of trunks by the emigrants in the *New York*, to obtain warmer clothing; but in the *Swanton*, as there had been no cold weather, this was unnecessary, and the baggage was undisturbed; yet the writer describing these occurrences in the *Cholera Epidemic of* 1873 *in the United States*, at page 608, gives the following quotation regarding them: "It seems to me far more probable that the poison was lurking in the luggage of the passengers, and that the emergencies which required the opening of the unventilated trunks and packages let loose in each of these ships that poison brought from the infected regions of Germany." The remark of the captain of the *Swanton*, that it was more like artificially heated air than anything else, affords a clue to the mystery that now enables it to be explained. Anyone who has experienced a hot wind, with which many of you no doubt are familiar, will see at once this is what he described, but, as a hot wind always originates over arid land, how could such a wind have been experienced from the south-east at the place where the *Swanton* was, from which a south-easterly line would pass through the South Atlantic clear of all land? The wind was really from the desert in North Africa. In November, when the north-east trade is being re-established over the Northern Atlantic, it reaches to 6 deg. N.; and about long. 30 deg. W., and lat. 10 deg. to 20 deg. N., at this season, north-easterly winds are frequently experienced such as are described by the captain of the *Swanton*, and, in addition, often bearing red dust in such quantity as to cover the sails and rigging of passing

vessels, leaving no doubt as to their place of origin, and further illustrating their transporting powers. Such a current of air, which did not come to the surface of the sea, but continued to flow at some elevation above it, would retain its peculiarities for a long time; it would pass on to the inner limit of the trade, about 6 deg. N. in November, and then rising in the atmosphere, as the air of the trade does, would double back to the north-west, constituting a south-east wind when it fell to the earth's surface, as the *Swanton* experienced it. In November 1848, when these occurrences took place, there had been cholera in Egypt and along the north coast of Africa, and, with a moderate velocity of twenty miles an hour, the wind could have transported emanations from that to the *Swanton* in from ten to twelve days.

The *New York* reached her destination on 1st December, having lost seven of her passengers and sent eleven to hospital, which number was increased to twenty the following day. Up to 28th December about 100 cases had occurred in quarantine, fifty of them fatal; and only two in the city. Though upwards of 100 of the passengers by the *New York* had absconded from the lazaret into New York, no more cases presented themselves in it until 11th May. The *Swanton* reached New Orleans on 11th December, having lost thirteen passengers from cholera. She went at once to her wharf, and proceeded to land her passengers and cargo. She sent one emigrant to hospital with cholera on arrival, and another the following day.* So rapid was the spread of the disease that, on the 16th December, it had diffused itself over a large portion of the city, and attacked those who could not, by any possibility, have had any communication with the ship or with each other. Dr. Fenner had attended, as early as 5th December, a gentleman who had symptoms of cholera so well marked that he should have pronounced it, but for the absence of the epidemic, a case of that disease. Cases of a similar

* It is but proper to notice here that Dr. Fenner mentioned subsequently, that, on December 6th, 1848, the ship *Gutenburg* arrived at New Orleans from Hamburg, with 250 German emigrants, after a passage of 55 days, during which six or seven deaths had occurred from cholera; one of the early cases at New Orleans was a man from this vessel. The barque *Callao* arrived from Bremen on December 8th, with 152 German emigrants, after a passage of 48 days, during which she had lost eighteen from cholera. The *Callao* was kept at Slaughterhouse Point, opposite to New Orleans, until January 4th, when she was brought over to the city and unloaded. (*Cholera Epidemic of* 1873 *in United States*, p. 610.)

kind occurred in the practice of physicians in different parts of the city, showing *that the epidemic influence of cholera was gradually being matured and developed.**

The following description, of the suffering of a body of United States troops, has an important connection with the first manifestation of cholera at New Orleans in 1848. The 8th Infantry, U.S.A., left Jefferson Barracks, near St. Louis on the Mississippi, on 24th November; they reached New Orleans on 1st December, and were landed at Jackson Barracks, three miles and a half below the city, and accommodated in sheds till the 12th, when they embarked in steamers for Lavacca, in Texas, and arrived there on the 14th. The disembarkation was not completed till the 19th December, when they were encamped on a brackish bayou or creek. On the 21st a wing was advanced about twelve miles, to a more favourable locality; that night a violent cold northerly wind came on, and "cholera showed itself in both camps, and, by the morning of the 22nd, had attacked more than one-eighth of both commands. The disease continued with great violence during the 22nd, 23rd, 24th, and 25th, when it began to abate; by the 30th it had entirely subsided." There were 133 deaths out of 196 cases (*Sickness and Mortality in the Army of the United States, Washington*, p. 398). These troops could not have been affected by the sick either from the *Gutenburg* or *Swanton*, yet the extension of the epidemic influence, and the depressing effects of the cold wind, and possibly to some extent the brackish water, caused a frightful mortality amongst them.

Another ship, the *Apollo*, which embarked the 59th Regiment (amounting, with women and children, to 693 persons) at Cork, for China, a few months after the *Swanton* and *New York*, suffered from cholera in her passage southwards, and affords an additional proof that a cholerific influence was, about that time, in operation extensively over the Atlantic. This ship was frigate-built, that is, with a gun-deck completely covered, but with the usual side-ports, and a lower deck called the orlop, with small side-scuttles only; the crew and part of the troops occupied the gun-deck, and the remainder of the troops, with the females and children, were accommodated on the orlop, the ventilation of which was naturally much inferior to that of the gun-deck. The troops embarked on the 12th June 1859,

* *Report of General Board of Health on the Epidemic Cholera of* 1848 *and* 1849, Appendix C, being Abstract of Report on Epidemic Cholera as it prevailed in the United States in 1849 and 1850, p. 79.

and the ship sailed on the 17th. Cholera had prevailed at Cork for some time, but there had been none in the barracks which the regiment had occupied before going on board, nor in the ship herself. On the morning of the 18th, a soldier, who had had diarrhœa for some days, got out of his hammock, much heated, and, placing himself under the tap, allowed the water to run over his body. Collapse ensued immediately; there were slight cramps, with the characteristic evacuations, and he died in seven hours. No other case occurred till 27th June, when a man was attacked in 35 deg. 21 min. N. and 15 deg. 25 min. W.; and up to July 1st, when the ship reached Teneriffe, another man, a woman, and two children had been attacked, all of whom died. From 2nd July to 15th, five men and a woman were attacked, all of whom recovered. On the 16th another man was attacked, who died. On the 17th, in 5 deg. 14 min. N., 24 deg. 3 min. W., there was heavy rain; and from the 18th to the 23rd there were fifteen men attacked, eight of them being part of the crew; seven of the fifteen died. The south-east trade commenced on the 24th, in 0 deg. 26 min. S., and subsequently to the 12th August seven other cases occurred among the troops, of which six proved fatal. The ship was now off Rio Janeiro, but, being refused pratique, proceeded to the Ilha Grande, sixty miles to the westward, where the troops and crew were landed, all her stores removed, and she was thoroughly disinfected. After this the crew and troops were re-embarked, and she completed her voyage to Hong Kong without any further trace of cholera. There had been much choleraic diarrhœa during the passage to Brazil, many of the cases so severe that the medical officer, Dr. Thomas Fraser (now Deputy Inspector-General on the retired list), was frequently in doubt whether they should not be classed as cholera, in addition to those so returned above.

To show that the outbreaks of cholera at New Orleans, and northern coast of the Gulf of Mexico, were not the only epidemics connected with the influence the *Swanton* and *Apollo* encountered in the middle of the Atlantic, it is to be noted that cholera appeared sporadically on the coast of New Granada, in South America, in June 1849, and became epidemic in the towns along the coast in July and August, and ascended the valley of the Magdalena to Bogota, about 500 miles inland and 8,000 feet above the sea. In Jamaica, at this time also, there were several sporadic cases of the malignant form, viz., a soldier of the 2nd West India Regiment, at Port Royal, in August, and,

in Kingston Barracks, some soldiers, as well as several civilians in the town, were affected about the same time. There was also a European soldier, quartered at the mountain-station Newcastle, who was attacked in October. The disease was much reduced in frequency on the coast of New Granada towards the end of 1849; but in the spring of 1850 it became active again, and extended westward. It became epidemic in Jamaica also, commencing on 7th October, and proved very destructive in that island. In 1850 there were four European soldiers attacked with cholera in Barbadoes from July to September, and one in St. Vincent, of whom three died; and ten more from October to December, at the former of which one died. Two white soldiers were also affected with cholera in Dominica in the last quarter of the year. There were also several cases of common cholera among the civil population at most of the Windward and Leeward Islands in 1850, a few of them proving fatal.

Hirsch gives the following case, in which the ship passed over nearly the same course as the *Apollo*, and presented incidents closely corresponding with those which that ship underwent:—

"On the 13th October 1855, the emigrant ship *Franzisca* sailed from Hamburg for Rio Janeiro, with 220 'tween deck passengers, thirteen officers and cabin passengers, and sixteen of crew. On the 23rd of October, or ten days from the sailing of the ship, a case of cholera, ending fatally, occurred in a man twenty-seven years old; a second case followed on the 8th of November, the ship being then in the latitude of Madeira; and now there developed an epidemic which lasted until the ship arrived in the harbour of Rio, on the 12th December, or over a period of nearly six weeks, although there were only sixteen deaths. Among the cabin passengers, officers, and crew not a single case of sickness occurred." (*Geographical and Historical Pathology*, vol. i, p. 486.) There was much cholera along the south coast of the Mediterranean from Egypt to Morocco in 1854-55; Madeira was affected in 1855; and Fogo, one of the Cape de Verde Islands, became affected in the beginning of July. A Sardinian vessel from Savona, with emigrants for Buenos Ayres, among whom there had been considerable sickness, arrived at Fogo on 30th June; the nature of their complaints seems not to have been inquired for, and the vessel was admitted to pratique and her passengers landed, and the vessel cleaned and disinfected. On 4th July five persons were attacked with cholera, and the

following day the deaths were so numerous that the authorities and principal people fled in terror to the next island, St. Jago. The population of Fogo at this time was 5,195, and the deaths from cholera were 123. In 1856, St. Vincent, another of the Cape de Verde group, became affected with cholera on 23rd August, which lasted twenty-eight days, during which period the deaths were 626 out of a population of 1,350. Consul Miller, who was there during the whole epidemic, was of opinion the epidemic was generated on the island, as the vessels from infected ports had brought no cases of sickness with them. (*Abstract of Information on Laws of Quarantine*, published by Board of Trade, Parliamentary Papers, 18th August 1860, p. 13.)

Cholera was prevalent in New Granada in 1854, and in Venezuela in 1855, but up to this year it had not appeared in Brazil; now, however, it broke out at Para in the middle of May, at Villa Imperatriz and Province of Amazona in the beginning of June, and at Bahia in the end of June. Shortly afterwards Rio Janeiro was affected, and the whole coast of Brazil from Rio Grande do Sul to the Amazon was under the disease. In November the epidemic died out everywhere in Brazil; though it still continued in Guiana. In this, as in the preceding instances, the activity of the causes of cholera over the sea at the same time as at Fogo and on the coast of Brazil, is distinctly marked.

In 1866, when cholera was extending over Western Europe and this country, four steam-ships with emigrants left Liverpool for America. These were the *England*, which sailed on 28th March, the *Virginia*, which sailed on 4th May, the *Union*, which sailed on 12th May, and the *Peruvian*, which left Liverpool about the same time as the *Union*. The cholera had not commenced at Liverpool when these vessels left, and none of their passengers were known to be affected; but when they reached a point in the Atlantic close to 48 deg. 50 min. N., and 28 deg. 40 min. W., the disease broke out in each, and she carried it to her destination. About this time there was an unusual number of indications of cholera in ships at sea, extending nearly as far south as the Cape of Good Hope, and a fatal case of it in Cape Town itself. Thus the *Renown*, which left Gibraltar with troops for the Cape on 21st August 1865, had two attacks of cholera, and one death on 5th September, in 12 deg. N., 27 deg. W.; and five days after, in 6 deg. N., 22 deg. W., another outbreak commenced, which lasted till 19th September, during which there were fifteen attacks,

twelve of which proved fatal. The ship's position on the 19th was 14 deg. S. and 30 deg. W. The following troops, viz., 357 officers and men, and seventy women and children, embarked in the *Windsor Castle* at Gravesend on 11th July 1866, and sailed the following day for Kurrachee. A young man, who was embarked on 11th from the cells, drank three or four pints of porter before going on board; he was attacked with cholera on the 12th, but recovered. The first attack at sea was on the 18th, which proved fatal. On 22nd, diarrhœa increased remarkably, and a soldier was affected with cholera, who recovered. From the 29th fresh cases of cholera occurred, the ship being then about 32 deg. N., 19 deg. W., until the 20th August, when she was in 12 deg. S., 25 deg. W.; altogether, four men, two women, and two children died of cholera; the number of attacks was not given. The last case at this time occurred on 20th August, and died on 3rd October, in 34 deg. S. and 8 deg. W., from fever and hæmorrhage. A sailor of the crew died of a fresh attack of cholera on 25th September, when to the east of the Cape, in 37 deg. S., 39 deg. E. The *Lord Warden* left Portsmouth for India on 23rd September 1866, with 306 officers and men, and 61 women and children, besides the crew, and some other passengers. On 25th September the first case of cholera occurred, which recovered. On 26th there was another, which died. Up to 7th October a few cases of diarrhœa presented themselves, and from the 8th to 24th there were nine cases of cholera, all fatal. There were fifteen attacks in all, of which ten proved fatal. The names of the sick and dates of attack are not given. The ship's position on 8th October was 36 deg. N., 17 deg. W., and on the 24th, when the last case died, 8 deg. N., 21 deg. W. The *Jumna*, one of the new Indian steam troop-ships, was on her passage outwards in 1867. She had besides her crew a number of military officers on board, but no troops. On 17th July, four days after leaving St. Vincent, one of the Cape de Verde Islands, one case of cholera and six of choleraic diarrhœa occurred among the crew, and the diarrhœa continued to present itself till the 27th, by which date forty-six had been entered on the sick-list, besides some ten or twelve milder which were not. The ship's position on the 17th, at noon, was 6 deg. 35 min. N., 15 deg. 46 min. W., and on the 27th 19 deg. 37 min. S., 5 deg. 27 min. W. Some fresh beef obtained in England thirteen days before, and brought out in the ice-house, was issued to the crew on the 17th; the Marines, who used the beef, suffered more than the rest of

the crew, while the warrant officers, who refused it, escaped. One of the military officers had a severe attack of choleraic diarrhœa. There was no cholera at St. Vincent when the *Jumna* was there. H.M.S. *Vestal* had one of her crew attacked with cholera on 21st May 1867, as she was entering the Gambia. The ship *Newcastle*, proceeding to England, had a case of cholera midway between the Cape and St. Helena, in April 1866; and in Cape Town itself, a lady was attacked with cholera in October, collapse ensued, and she died with the characteristic symptoms of the disease.

While the manifestations just described were occurring along the eastern shores of the Atlantic, and in the vessels traversing it, cholera appeared in November 1865 at Guadaloupe, in the West Indies, and slighter indications of the same were met with in Dominica and Martinique. In Brazil, where cholera had not been met with since 1856, it again broke out, not on the sea-coast, but far inland at Corrientes, the point of junction of the rivers Parana and Paraguay, where the armies of Brazil and the Argentine Republic were watching that of Paraguay. The cholera appeared first in April 1866, in the army of Paraguay, but soon extended to the opposing forces, and to places in the neighbourhood; but it was thought it had been prevented extending to Buenos Ayres by strict quarantine, perhaps also by the intervention of the cool season. In January 1867 it commenced again and descended the river; but though there were slight manifestations of it at Buenos Ayres in December, the active progress of the epidemic was again stopped during the cool weather. In the summer 1867-68, however, it revived and attacked Monte Video, and extended afresh over the whole area that had already been affected by it, as well as much of the Monte Videan territory. It is difficult in the present state of the information regarding these varied outbreaks to analyse the facts so as to bring out completely the active causes, but it is obvious from what has been advanced above that the generally accepted reasoning, regarding causes and mode of propagation of cholera, require profound modification.

THE SEASONAL PREVALENCE OF ENTERIC FEVER.—(*Abstract.*)

By LOUIS PARKES, M.D.Lond., D.P.H., Lecturer on Public Health at St. George's Hospital, Medical Officer of Health for Chelsea.

(*Read: May 18th*, 1892.)

THE reduction in the death-rate from enteric fever in London from 337 per 100,000 in the earlier to 150 to 160 in the later years of the periods 1870 to 1890, that is, since it had been officially distinguished from other forms of "fever" observed throughout the country, was doubtless due no less to better domestic sanitation, especially in the dwellings of the poor, than to improved water-supply and sewerage, which had been for some years previously practically the same as since. Dr. Parkes stated that he would confine his observation to London, the enormous population of which minimised the disturbing effects of local outbreaks due to accidental causes, and where the largest aggregation of persons living under nearly identical conditions was to be found; his purpose being to inquire into the relations, if any, between the greater or less prevalence of the fever and the special meteorological conditions and rainfalls of the different years. Enteric fever showed so constant a seasonal curve that it was easy to determine the average number of deaths each week over a long series of years, and drawing a base-line for the mean, to represent the percentage of weekly excess or defect, while the duration of the disease and of the incubation being known, one could infer the period of greatest activity. The curve, as drawn by Buchan and Mitchell, showed a rise from its lowest point at the end of June, crossing the mean in August, suddenly rising in October to its maximum, falling slowly in November, rapidly in December, recrossing the mean line in January, and, with the exception of short rises in February and March, sinking continuously till the end of June, the highest point being 40 per cent. above and the lowest 30 per cent. below the mean. A temperature above 60 deg. F. being as a rule necessary for the development of

the highest degree of virulence in the enteric fever bacillus, whether in the soil or in water, it was but natural to expect that, as Murchison had maintained, the hottest seasons would be the years of greatest and the coldest of least prevalence. Hot seasons were mostly dry, and wet ones, cold; but heavy rains, the weather being warm, might pollute wells in rural districts or flush the sewers in towns, which would explain the discrepancies between the actual prevalences in London and the provinces, and the temperature in certain years. Giving three weeks for the incubation and the same for the duration of fatal cases, the greatest activity of enteric fever would be in September. The highest temperature of the soil was indeed reached some three weeks earlier, but the interval represented the period of development and general diffusion of the bacilli, which retained their activity for some months after the temperature had begun to fall, the effect of cold being far less marked on enteric fever than on diarrhœa. A series of tables was shown in which the years were arranged in order of the temperature, the rainfall, and the prevalence of fever in the last twenty weeks of each; and another in which they were grouped as A, years comforming well to the rule which asserted a relation between the prevalence of enteric fever in the autumn and the temperature and rainfall of the preceding summer; B, those conforming approximately to this rule; and, C, those not conforming thereto, either (1) of low summer temperature but much autumnal fever, and (2) of high temperature and little fever. Under A were included 1887, 1873, and 1888; under B, 1877, 1886, 1874, 1880, 1890, 1875, 1882, 1876, and 1885. C (1) included 1881, 1889, 1883, 1871, and 1879; and C (2), 1870, 1878, 1884, and 1872. The ascending curve crossed the mean line in July in three years, namely, 1870, 1884, and 1886, so that the autumnal excess of deaths was really greater than shown by the last twenty weeks only, and 1870 and 1874 were more conformable to the rule than they appeared to be, but the temperature of these summers was not exceptionally high. In eight years it crossed in August, in eight in September, and once early in October. No positive conclusions could be drawn from a series in which the exceptions were nine and the examples of the rule eleven; but this was probably explained by the equability of our climate, the mean summer temperature of the hottest year, 1887, having been only 5 deg. F. above that of the coldest, 1879, and its maximum 8.6 deg. above the mean of the maxima. Even 1881, with the

enormous mortality of 74 per cent. above the mean of the year, was a year of average temperature, and with a rainfall for that period of only one inch in excess. The differences in the rainfalls were much greater, but did not seem to exert any marked effect on the mortality. There were therefore other influences to be investigated, some favourable and others antagonistic, which in this country served to conceal the influence, however real, of temperature and rainfall on the seasonal prevalence of enteric fever.

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